CLINICAL STUDY, EVALUATION AND MANAGEMENT OF UPPER GASTROINTESTINAL BLEEDING

A DISSERTATION SUBMITTED TO

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In partial fulfilment of the regulations for the award of the

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DEPARTMENT OF GENERAL SURGERY

STANLEY MEDICAL COLLEGE AND HOSPITAL

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY CHENNAI

APRIL 2013

CERTIFICATE

This is to certify that the dissertation entitled **"Clinical Study, Evaluation and Management of Upper Gastrointestinal Bleeding"** is the bonafide work done by **Dr. K.S.Saravana Krushna Raja.,** Post Graduate student (2010 – 2013) in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfilment of the regulations of The Tamil Nadu Dr. M.G.R Medical University, Chennai for the award of M.S., Degree (General Surgery) Branch - I, Examination to be held in April 2013.

Prof. A. RAJENDRAN, M.S.,
Professor of Surgery,
Dept. of General Surgery,
Stanley Medical College,
Chennai-600001.

Prof. P. DARWIN M.S.
Professor and Head of surgery,
Dept. of General Surgery,
Stanley Medical College,
Chennai-600001.

PROF. S. GEETHA LAKSHMI, M.D., PhD,
The Dean,
Stanley Medical College,
Chennai-600001.

DECLARATION

I, solemnly declare that this dissertation titled "**Clinical Study, Evaluation and Management of Upper Gastrointestinal Bleeding**" is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of my unit chief **Prof. A. RAJENDRAN, M.S.,** and my Head of the Department **Prof. P. DARWIN, M.S.**

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the university regulations for the award of M.S., Degree (General Surgery) Branch - I, Examination to be held in April 2013.

Place : Chennai

Date : December 2012. **DR. K. S. SARAVANA KRUSHNA RAJA**

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CONTENTS

INTRODUCTION

Upper Gastrointestinal bleeding is a common potentially life threatening condition associated with high morbidity, mortality and medical care costs. Clinically manifests as haematemesis and, or melena and rarely haematochezia with or without haemodynamic compromise.

Upper Gastrointestinal bleeding is defined as bleeding proximal to the ligament of Treitz. The incidence of UGI bleeding is approximately 100 cases per 100,000 population per year. Mortality rates from UGI bleeding are 6 – 10 % overall[1]. Accurate patient evaluation and appropriate early management is critical to decrease the morbidity and mortality. The foundation of diagnosis and management of patients with Upper GI Bleeding is Oesophago-Gastro-Duodenoscopy (OGD). Endoscopy has a sensitivity of 92% for identification of the site of (AUGIB), with a specificity that approaches 100% , especially if it is done within the first 24 hour of (AUGIB)[2] . Various scoring systems have been used in the prediction of risk in patients with Upper GI bleeding and early stratification in accordance with clinical symptoms. Non variceal bleeding is due to arterial haemorrhage such as ulcers and deep mucosal tears, where as swollen veins due to portal

hypertension cause variceal bleeding and should be managed accordingly[3].

AIMS AND OBJECTIVES

1. This study reviews the clinical presentation , diagnostic modalities and evaluation of Upper GI bleeding.

2. To analyse the incidence and severity of presentation.

3. To predict the prognosis and mortality risk of Non variceal bleeding using Rockall scoring system.

4. To delineate the specific cause and bleeding site using OGD Scopy.

5. To analyse various modalities in the early resuscitation and management of Upper GI bleeding.

REVIEW OF LITERATURE

HISTORICAL ASPECTS

For more than 5000 years, upper Gastro Intestinal bleeding is one of the recognized causes of death. Various documents like Chinese manuscripts, Egyptian papyri, medical works of Hippocrates and not to forget famous Indian Surgeon,Sushruta, all have mentioned the upper gastrointestinal bleeding(non variceal) as one of the conditions associated with very high mortality. During ancient times, it was gradually brought to notice by symptomatology of the patients[4]. There were lot of evidences regarding the incidence of peptic ulcer were studied since from first century [5] .Morgagni in 1700 was the first person to describe the gastrointestinal bleeding because of portal hypertension[6]. Only at the turn of the 20th century with advent of endoscopy, evidence of haematemesis due to rupture of esophageal varices has been well established. Most of the earlier pioneers in esophageal endoscopic procedures including Crafoord used rigid endoscope (Negus Type). Now the flexible fibreoptic endoscope(Introduced in 1980) has replaced the rigid scope in almost all centres, which is supported by the result of controlled trial from Capetown. Endoscopy is now 50 years old and has established strongly it’s place in diagnosis and treatment of disorders of upper gastrointestinal tract both in

emergency and non emergency situations. Crafoord and Frenckner from Sweden were first to use sclerotherapy in 1936 to treat esophagealvarices in a 19 year old patient. In 1990, Stiegmann published the ligating device using rubber or latex for varices. Spices and herbs were used in ancient times to treat Peptic ulcer disease. The changes in our management of gastrointestinal bleedingoverthecenturieshavebeendrivenbynaturalalterationsinthespectrum ofdiseases,expanding our understanding of these diseases and the never ending advances in technology and pharmacology that have occurred relative to Upper Gastrointestinal diseases.

ANATOMY

OESOPHAGUS

Oesophagus is a fibro muscular tube which extends from cricoid Cartilage (C6, vertebra) to Oesophago gastric junction (T11, Vertebra)measuring 25cms. It is divided into cervical, thoracic and abdominal oesophagus. An anatomical sphincter is at the upper end of the oesophagus and physiological sphincter at the lower end of the oesophagus. In passing a Oesophago Gastro Duodenoscope, Cricopharyngeus sphincter can be identified which opens and closes intermittently.[7]

BLOOD SUPPLY

ARTERIAL SUPPLY

Inferior thyroid arteries:

The paired inferior thyroid arteries supplies the cervical esophagus that gives off branches called tracheoesophageal arteries

Tracheobronchial and Bronchoesophageal arteries:

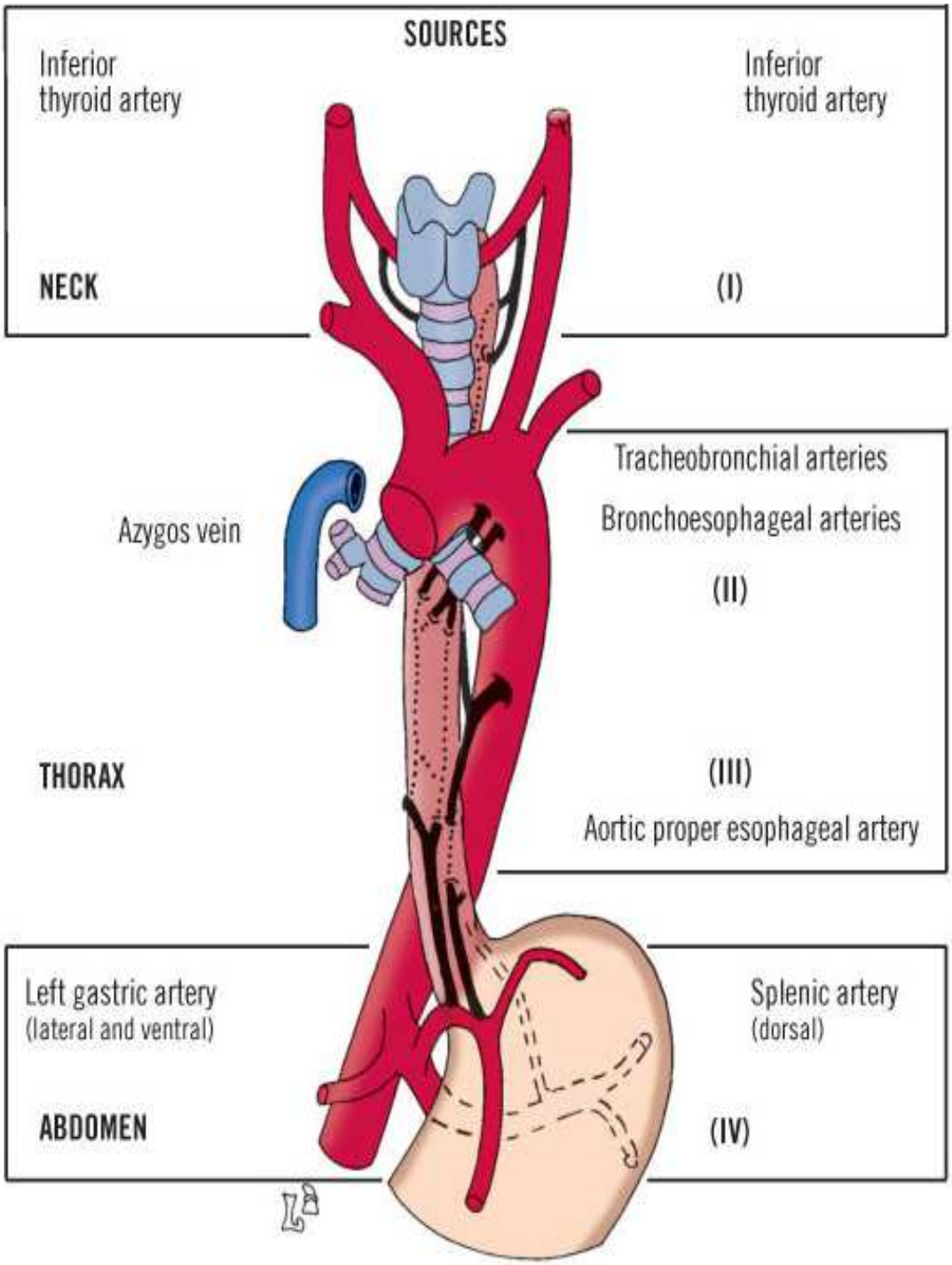
The tracheobronchial arteries give off multiple small branches to the esophagus which subdivide within the periesophageal tissue. Commonly, one bronchoesophageal artery originates 1 cm to 3 cm caudal to the vascular bundle from the anterolateral aspect of the descending aorta.

Aortic proper Oesophageal Artery:

It arises from the anterior aspect of the descending aorta.

Left Gastric and Splenic Arteries:

The left gastric artery mainly supplies the anterior and right lateral aspects of the esophageal wall. The splenic artery primarily supports the posterior and left lateral aspects (cardiac notch) by either one or two direct branches or by vessels of the gastric fundus, including connections with the short gastrics


SOURCES
Inferior thyroid artery
Inferior thyroid artery
NECK
(I)
Azygos vein
Tracheobronchial arteries
Bronchoesophageal arteries
(II)
THORAX
(III)
Aortic proper esophageal artery
Left gastric artery (lateral and ventral)
Splenic artery (dorsal)
ABDOMEN
(IV)

Venous Drainage:

The extrinsic veins drain into the locally corresponding large vessels. The superior vessels drain to the jugular veins or the azygos and hemiazygos veins. The inferior veins terminate in the left gastric and splenic veins.

STOMACH:

The stomach is divided into, Cardia, fundus, body and pylorus.

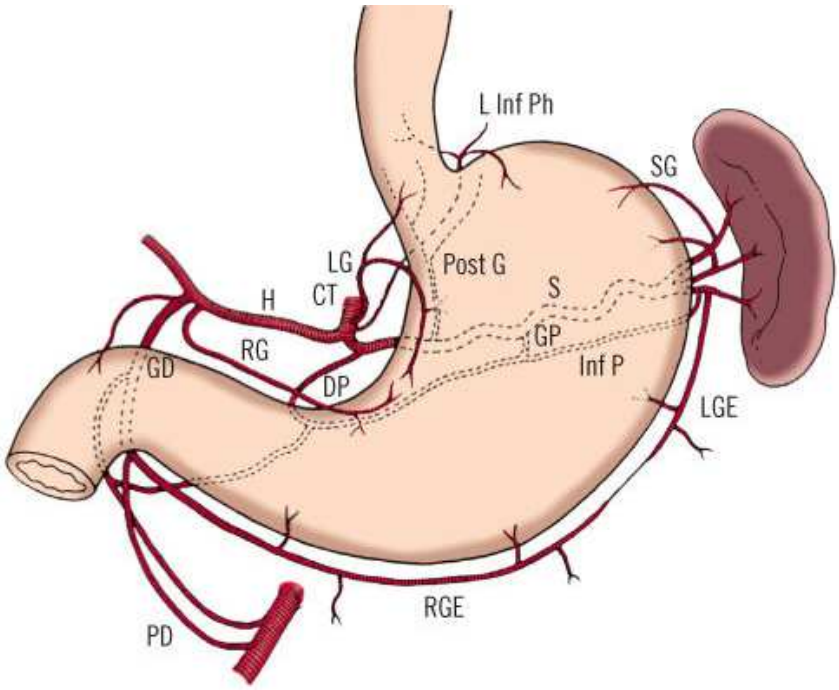
BLOOD SUPPLY:

ARTERIAL SUPPLY:

Left Gastric Artery:

In more than 90% of individuals, the left gastric artery is a branch of the celiac axis. The left gastric artery commonly divides into an anterior and a posterior branch before attaining the lesser curvature, it bifurcates into an anterior branch which sends branches to the anterior gastric wall, and a posterior branch which, similarly, supplies the posterior gastric wall. The anterior branch of the left gastric artery angles rather obliquely across the body of the stomach toward the greater curvature. It ends in numerous small ramifications and forms a vascular "crow's foot" (of Payne) and the posterior branch follows the lesser curvature a centimeter or two from its edge until it anastomoses with the right gastric artery The anterior and

posterior gastric branches may possess direct interconnections with one another or with the continuing segment of the parent left gastric artery.



Right Gastric Artery:

The right gastric artery is a small branch which arises most commonly from the proper hepatic artery(62%) but also arises from left hepatic artery, rarely from common hepatic.A. It gives origin to one or more suprapyloric branches.

Gastroduodenal Artery:

The gastroduodenal artery arises as one of the two terminal branches of the common hepatic artery. It gives origin to the supraduodenal, retroduodenal, and posterior superior pancreaticoduodenal arteries.

Right Gastorepiploic Artery:

The right gastroepiploic artery is a branch of the gastroduodenal artery (or its continuation) in most cases. After giving origin to an infrapyloric branch, the artery passes along the greater curvature of the distal gastric surgical unit within the gastrocolic ligament. The gastric branches of the right gastroepiploic artery pass mostly undivided in a submucosal position about one-fifth of the distance from the greater curvature. They anastomose extensively with branches from the left gastric artery.

Left Gastroepiploic Artery:

It arises in most cases (75%) from the distal splenic, inferior splenic terminal, middle part of the splenic trunk, or superior splenic terminal. It is the largest branch of the splenic artery gives off the left epiploic and the anterior epiploics.

Short Gastric Arteries:

Approximately five to seven short gastric arteries arise from the terminal branches of the splenic artery or from the left gastroepiploic artery.

Venous Drainage:

The venous supply of stomach usually accompany the corresponding arteries. A great venous arch can develop between the left and right gastroepiploic veins during portal hypertension, forming a congested vascular bridge between the splenic and portal veins.

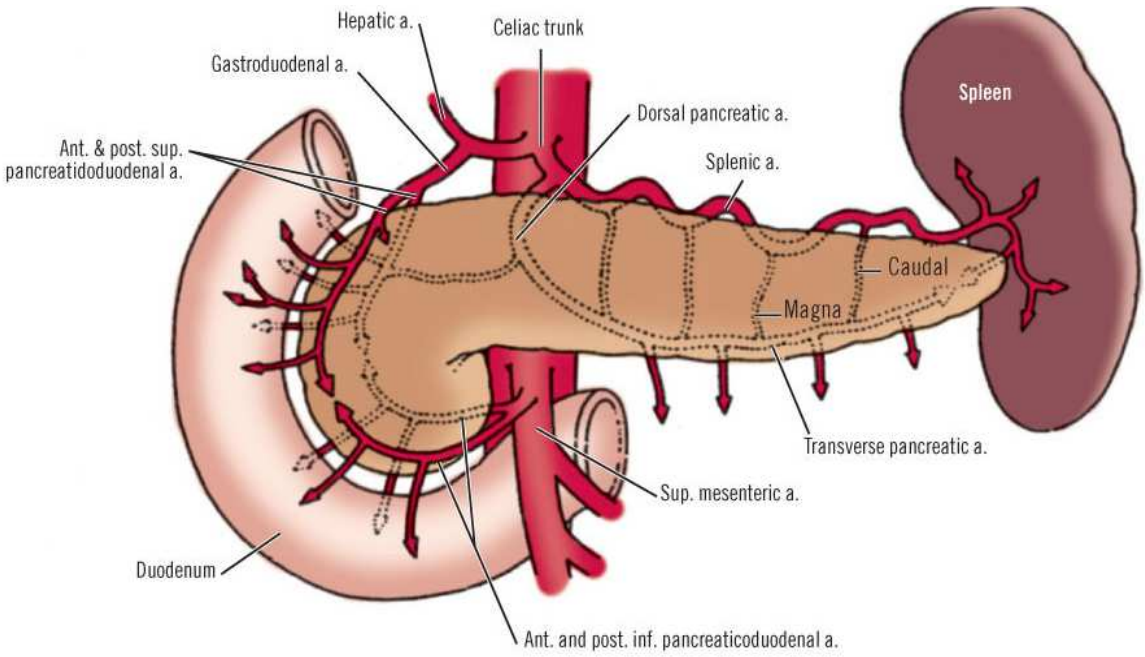
DUODENUM:

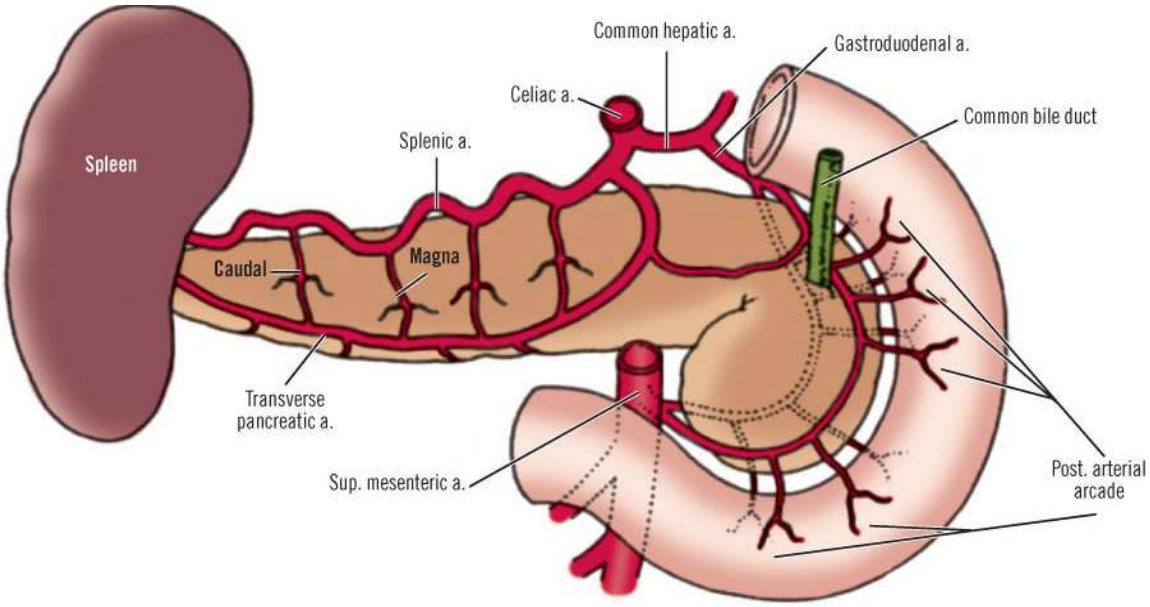
The duodenum has 4 parts:

The first (superior) part, or bulb (5 cm), The second (descending) part or C loop (10 cm),The third (horizontal) part (7.5 cm), The fourth (ascending) part (2.5 cm) continues into jejunum at duodenojejunal flexure.

Blood supply:

Arterial supply:





The first part of the duodenum is supplied by the supraduodenal artery and the postero superior pancreaticoduodenal branch of the gastroduodenal artery. The remaining three parts of the duodenum are supplied by an anterior and a posterior arcade.

Venous Drainage:

Veins of the lower first part of the duodenum and the pylorus usually open into the right gastroepiploic veins they are the subpyloric veins while the upper first part of the duodenum is drained by suprapyloric veins. The venous arcades draining the remaining duodenum follow the arterial arcades and tend to lie superficial to them.

PORTAL VEIN:

The superior mesenteric and splenic veins join posterior to the neck of the pancreas to form the main portal vein[9]. It receives pyloric and coronary vein branches as it courses cephalad and obliquely to the right to form the most posterior structure within the hepatoduodenal ligament (portal triad). In the hilum of the liver, the main portal vein bifurcates into a short oblique right portal vein and a longer, more transverse, and more superficial left portal vein[10] . These branches then enter the parenchyma

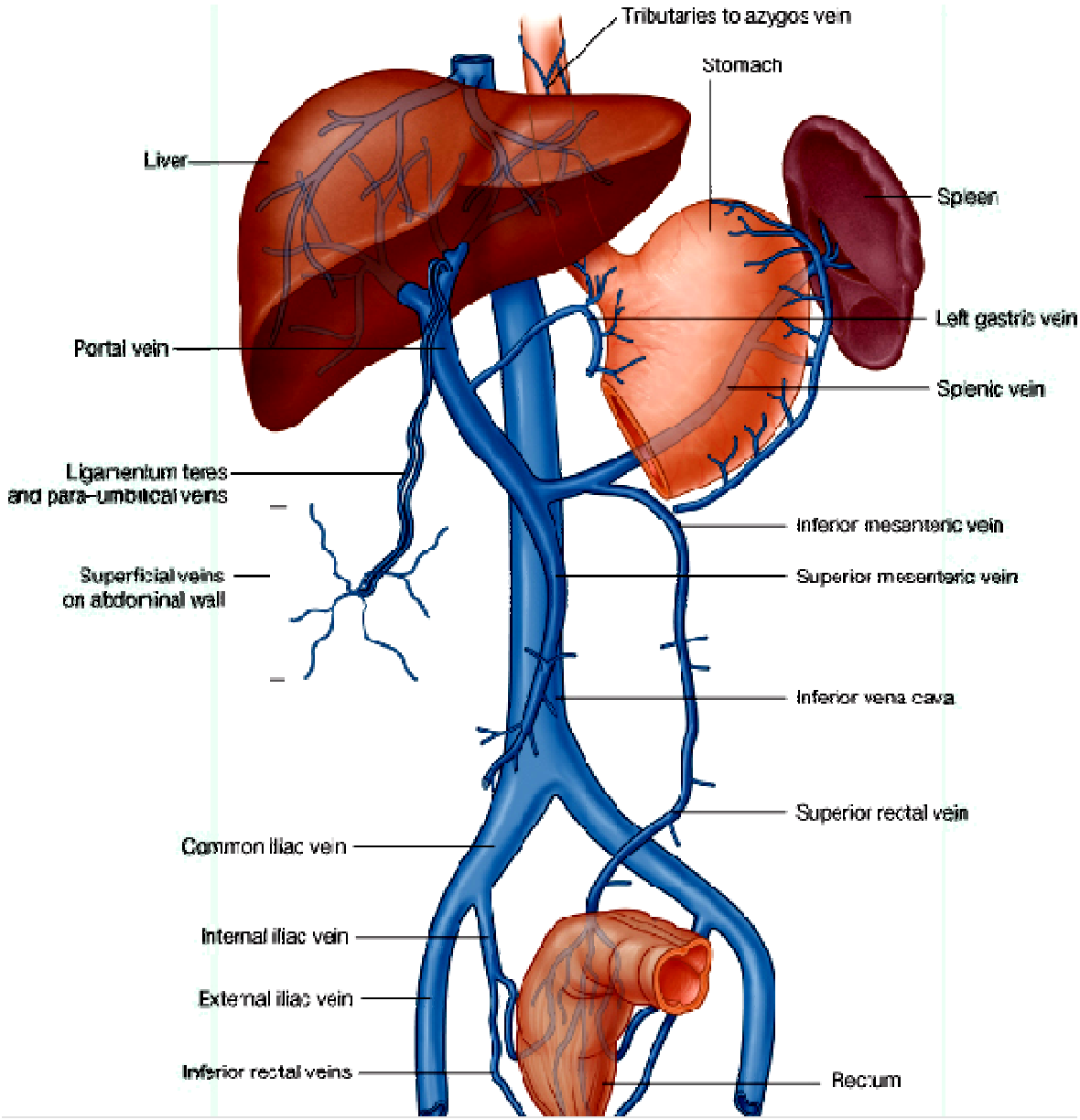
and become invested along with the other components of the portal triad by extensions of Glisson's capsule.

Porto systemic collaterals:

Multiple anastamoses forming between the portal and systemic circulation contributing the collateral circulation as follows,

- Lt gastric vein anastamose with oesophageal veins.
- Superior rectal vein with middle and inferior rectal veins.
- Para umbilical veins with anterior abdominal wall subcutaneous veins.
- Splenic and pancreatic vein tributaries anastomose with left renal vein retroperitoneal area[13].
- Veins over the bare area of liver anastomose with with the veins of the diaphragm and rt. internal thoracic vein[14].

PORTO SYSTEMIC COLLATERALS



EPIDEMIOLOGY:

The age distribution varies depending on the studied population affecting the elderly in the west [16,17]. The male:female ratio for (AUGIB) in many European countries and the United States is approximately 2:1 .There is regional variation regarding the frequency of causes of (AUGIB) depending on the demographic characteristics of the studied population, risk factors of bleeding, timing of the study and pathological classification[17].The incidence rates of UGIB demonstrates a large geographic variation ranging from 50 to 160 cases per100 000 population, with consistent reports of higher incidences among men and elderly people. Possible explanations for this reported geographic variation in incidence are differences in definition of UGIB in various studies, population characteristics, prevalence of alcoholism, ulcerogenic medication, in particular aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), and Helicobacter pylori(H. pylori) prevalence[18,21]. Acute variceal bleeding has a significant mortality which ranges from 5% to 50% in patients with cirrhosis[23]. Some studies have reported a significant decline in incidence of acute UGIB, especially peptic ulcer bleeding, in recent years[22,23].This decline is likely due to a combination of factors, including decreasing prevalence of gastric colonization with H. pylori, the use of eradication therapy in patients with ulcer disease, and the increased

use of PPI therapy,both in general and in patients using aspirin and NSAIDsin particular. Despite the introduction of therapeutic endoscopy and acid-suppressive therapy, the overall mortality of UGIB has remained stable over recent decades and is still 6%-14% in most studies[24] . As such, mortality from UGIB is strongly associated with advanced age and presence of severe comorbidity. The risk of mortality increases with rebleeding, which is thus another major outcome parameter. It was noticed that, mortality due to haemetemesis after admision was more than those who admitted with symptoms[25] . The incidence of rebleeding in patients with UGIB shows a wide range from 5% - 20%, depending on several factors. One of the prognostic index of rebleeding was increased portal pressure > 15 mmHg, and the same was also confirmed in a study of Moitinho et al[26].

More than 25 percent of episodes of UGI bleeding were due to pepti ulcers among 7800 individuals included in a database of USA between 1999 and 2001[27]. Nonspecific mucosal abnormalities appeared to be commonest(40 percent), while Oesophagitis in 12 percent, and Oesophageal and gastric varicesin about 10 percent. Other causes (AV malformations, Mallory-Weiss tears, and GI tumors) were seen in less than 5 percent of cases. Among the patients with peptic ulcer disease,

gastric ulcers were seen in more than fifty percent than duodenal ulcers[28].

In another study focused on OGD performedfor a period of 4 years from the year 2000 in a setting, the endoscopic findings that was common in patients with UGIB were Peptic ulcer (31 percent) followed by an Gastric or duodenal erosion (20 percent). Gastric ulcers in OGD were dominant than duodenal ulcers (54 vs 36 percent)[29].

Recent epidemiological studies had revealed a decrease in incidence of all causes of UGIB except those of peptic ulcer bleed. Bleeding due to varices seems be the leading cause of bleeding in cirrhotic patients in 50-60%[30]. Rebleeding in occurs in 10-16%, despite the therapeutic modalities. Mortality ranges between 3 to 12 % and does not pose a change in the last few years. NSAIDs was only used in 12% of the individuals who were presented with bleeding. H. pylori infection is found in about 45% of individuals with bleeding peptic ulcer. H. pylori should be kept in mind in all patients with peptic ulcer and eradication should be given.

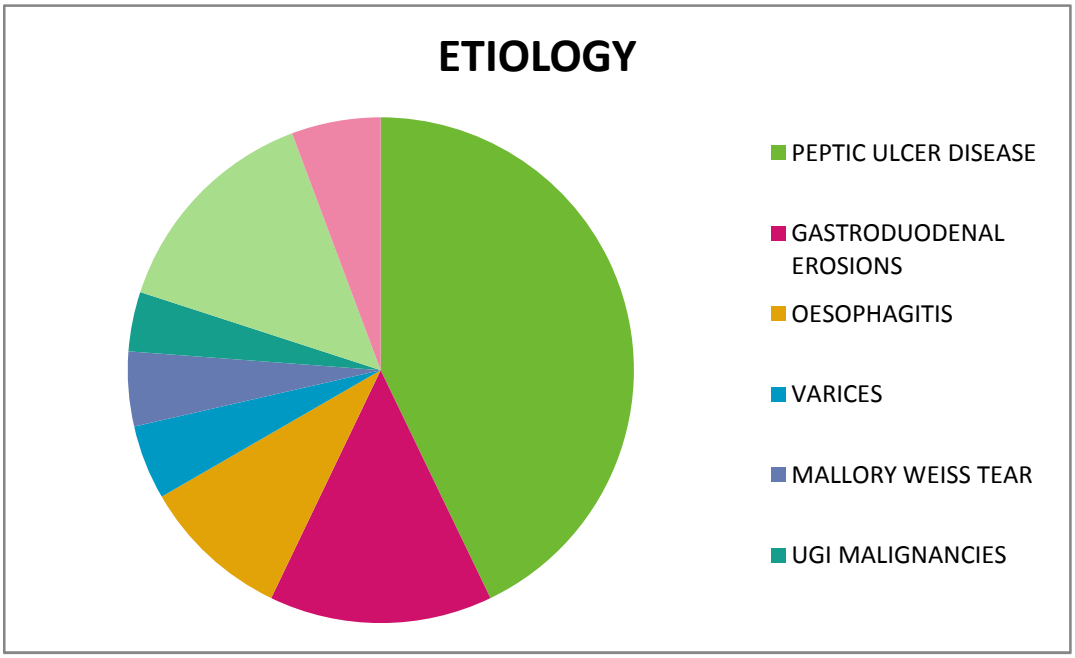
Child, in his classic monograph emphasized the co-existence of hepatic disease and manifestations of portal hypertension. It was only in the turn of twentieth Century that Gilbert and associates, and Pichancourt coined the term “portal hypertension”. At the same time several pathologists related

the formation of esophago gastric varices to portal hypertension[30].The incidence of varices from 5%to15% ofpatients with cirrhosis per annum.

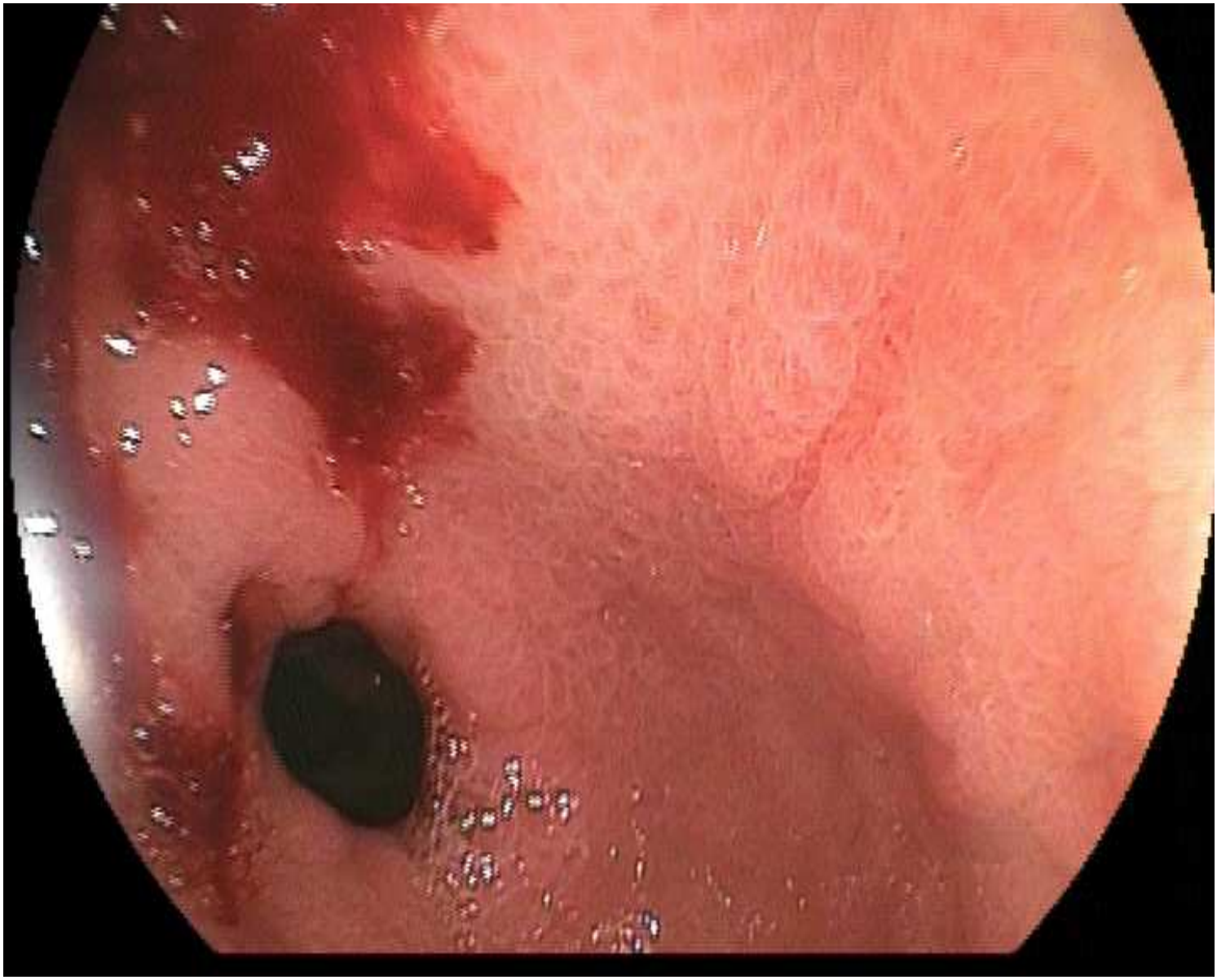
ETIOLOGICAL FACTORS:

According to various studies, the incidence of etiological factors were as shown below,

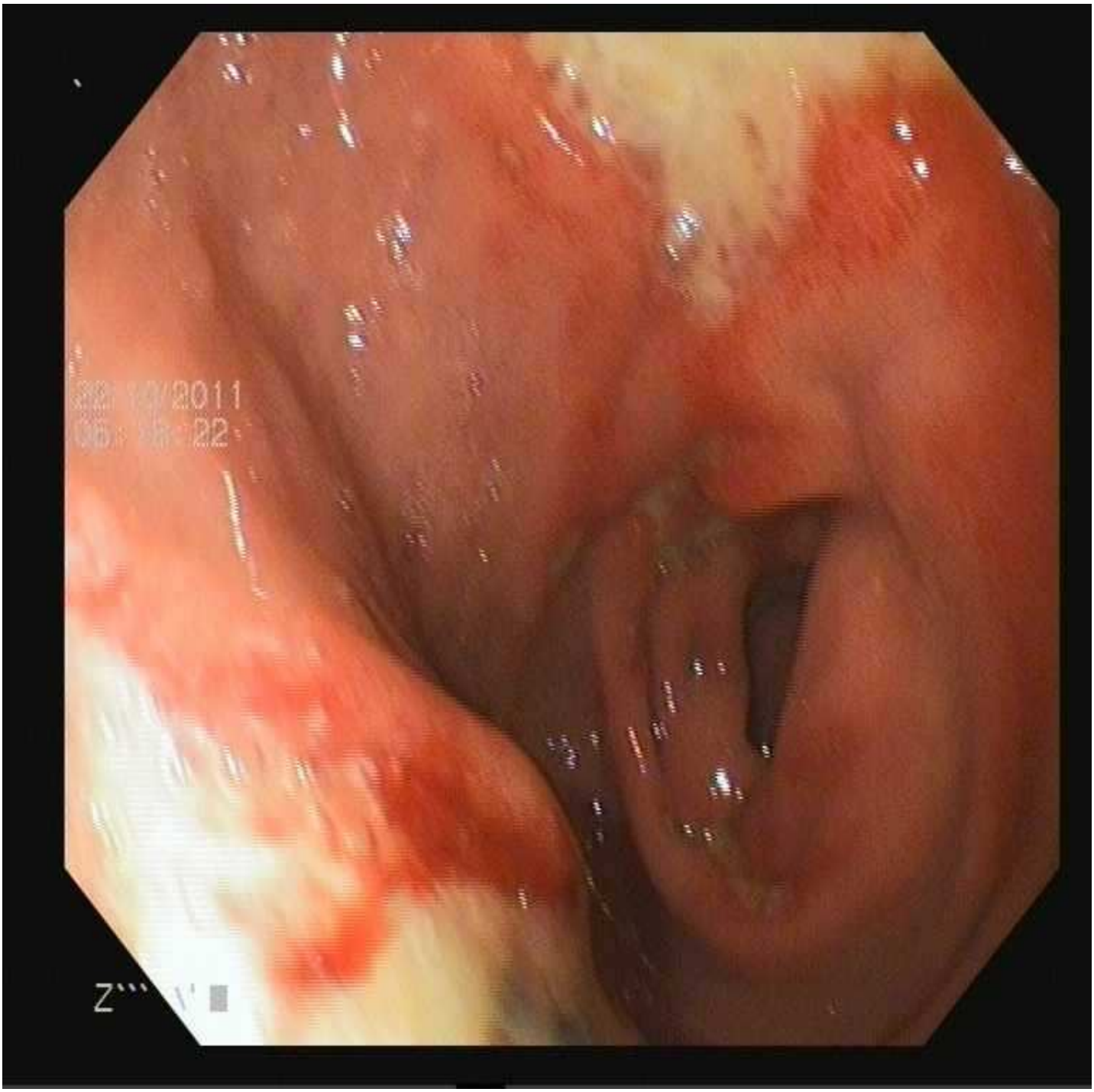
PEPTIC ULCER	40%
NO OBVIOUS CAUSE	15%
EROSIVE DISEASE	15%
OESOPHAGITIS	10%
OTHERS	6%
MALLORY WEISS TEAR	5%
VARICES	5%
NEOPLASM	4%



EROSIVE GASTRITIS



GASTRIC ULCER



Peptic ulcer disease:

Peptic ulcers are focal defects in the gastric or duodenal mucosa that extend into the submucosa or deeper to it. They may be acute or chronic caused by an imbalance between mucosal defenses and acid injury. Common sites for peptic ulcers are the 1st part of the duodenumand the lesser curve of the stomach, but they also occur on the stomal site following gastric surgery[32]. It remains as the most common cause of life threatening UGIB. Bleeding mainly occurs from the underlying arterial erosion and it depends upon the size of the erosion and diameter of the vessel. There are 5 types of gastric ulcer, classified depending upon the anatomical location. Among these types, Type I seems to be the common ulcer located near the incisura angularis of lesser curvature.

H.pylori is the most important factor in the development of peptic ulceration. About 3 /4 of duodenal ulcers and 1 /4 of gastric ulcers were caused by H.pylori infection. With the use of antibiotics, the prevalence of the infection have been much decreased in USA. The other factors associated with peptic ulcer formation are stress induced, NSAIDs intake, smoking and alcoholism. even after treatment with proton pump inhibitor therapy[33].

Mallory weiss tear:

These are tears seen at the oesophago gastric junction, that cause UGIB. Due to continuous vomiting or with retching, haemetemesis occurs and are commonly associated with alcoholism and chemotherapeutic agents like cisplatin etc[35].

Portal Hypertension:

The portal venous system contributes approximately 75% of the blood and 75% of the oxygen supplied to the liver[31]. In the average adult 1000 to 1500 mL/min of portal venous blood is supplied to the liver. However, this amount can be significantly increased in the cirrhotic patient. The portal venous system is without valves and drains blood from the spleen, pancreas, gallbladder, and abdominal portion of the alimentary tract into the liver[34]. Tributaries of the portal vein communicate with veins draining directly into the systemic circulation. These communications occur at the gastroesophageal junction, anal canal, falciform ligament, splenic venous bed and left renal vein, and retroperitoneum . The normal portal venous pressure is 5 to 10 mmHg, and at this pressure minimum blood is shunted from the portal venous system into the systemic circulation. As portal venous pressure increases, however, the communications with the systemic circulation dilate, and a large amount of

blood may be shunted around the liver and into the systemic circulation[37].

A WHVP or direct portal venous pressure that is >5 mmHg greater than the inferior vena cava (IVC) pressure, a splenic pressure of >15 mmHg, or a portal venous pressure measured at surgery of >20 mmHg is abnormal and indicates portal hypertension. A portal pressure of >12 mmHg is necessary for varices to form and subsequently bleed[38].

Depending on this, the etiological factors implicated in portal hypertension can be categorized into four major groups:

1. Increased Hepatic portal flow.
2. Extra Hepatic outflow obstruction.
3. Obstruction of the extrahepatic venous systems.
4. Intrahepatic obstruction.

90% of the cases with portal hypertension are due to intrahepatic obstruction.

Causes for portal hypertension:

I. Cardiac diseases

1. Right ventricular failure

2. Tricuspid stenosis

II. Vascular diseases:

1. Budd- chiari syndrome

2. Membranous obstruction of the Inferior Vena Cava

3. Thrombosis of the inferior vena cava.

III. Acute and chronic Liver diseases

1. Cirrhosis.

2. Idiopathic portal Hypertension.

3. Schistosomiasis.

4. Congenital hepatic fibrosis.

5. Exposure to environmental toxins.

6. Metastatic carcinoma.

IV. Venous Occlusion of Portal System.

1. Portal Vein

2. Splenic Vein

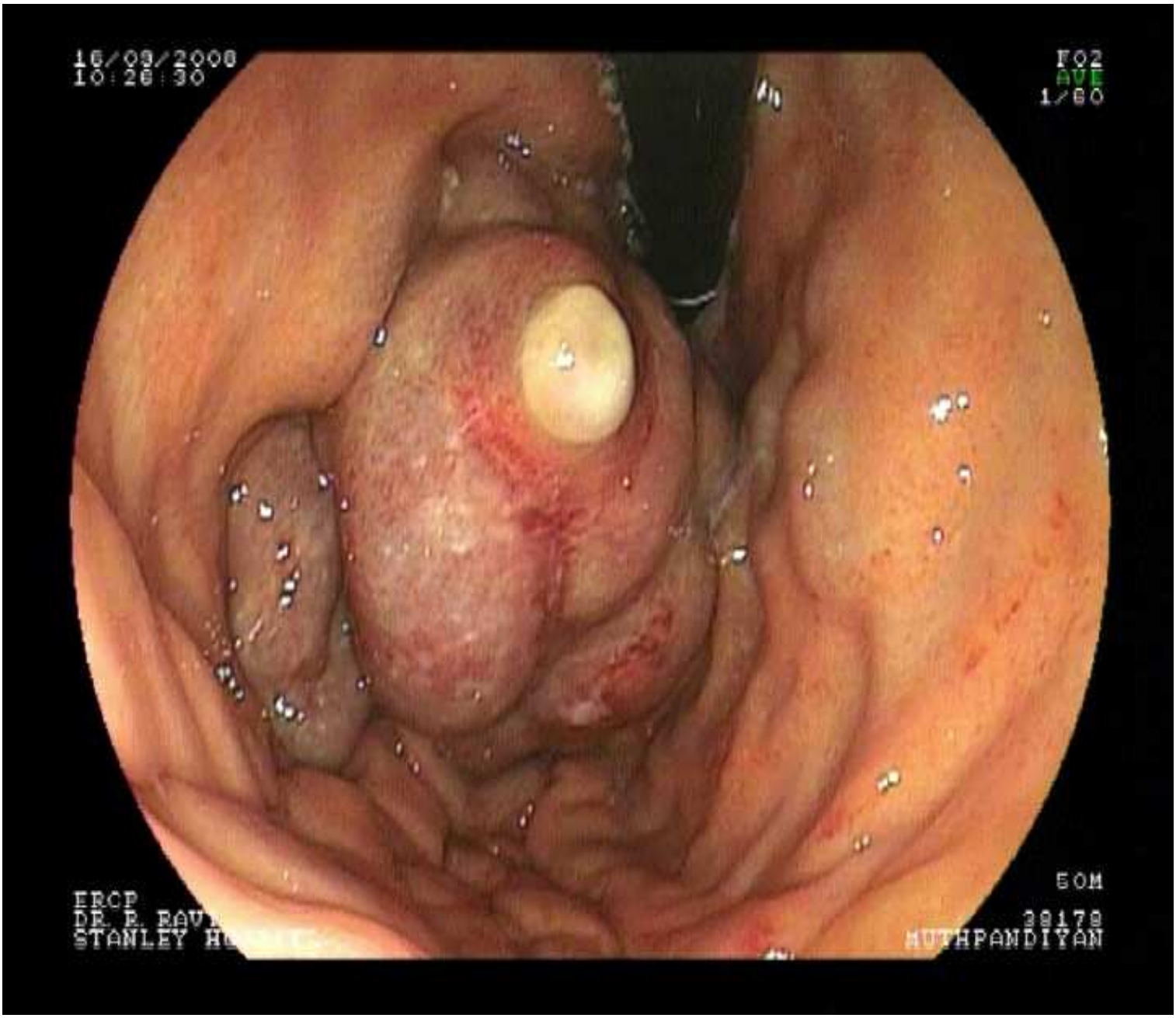
Oesophageal Varices

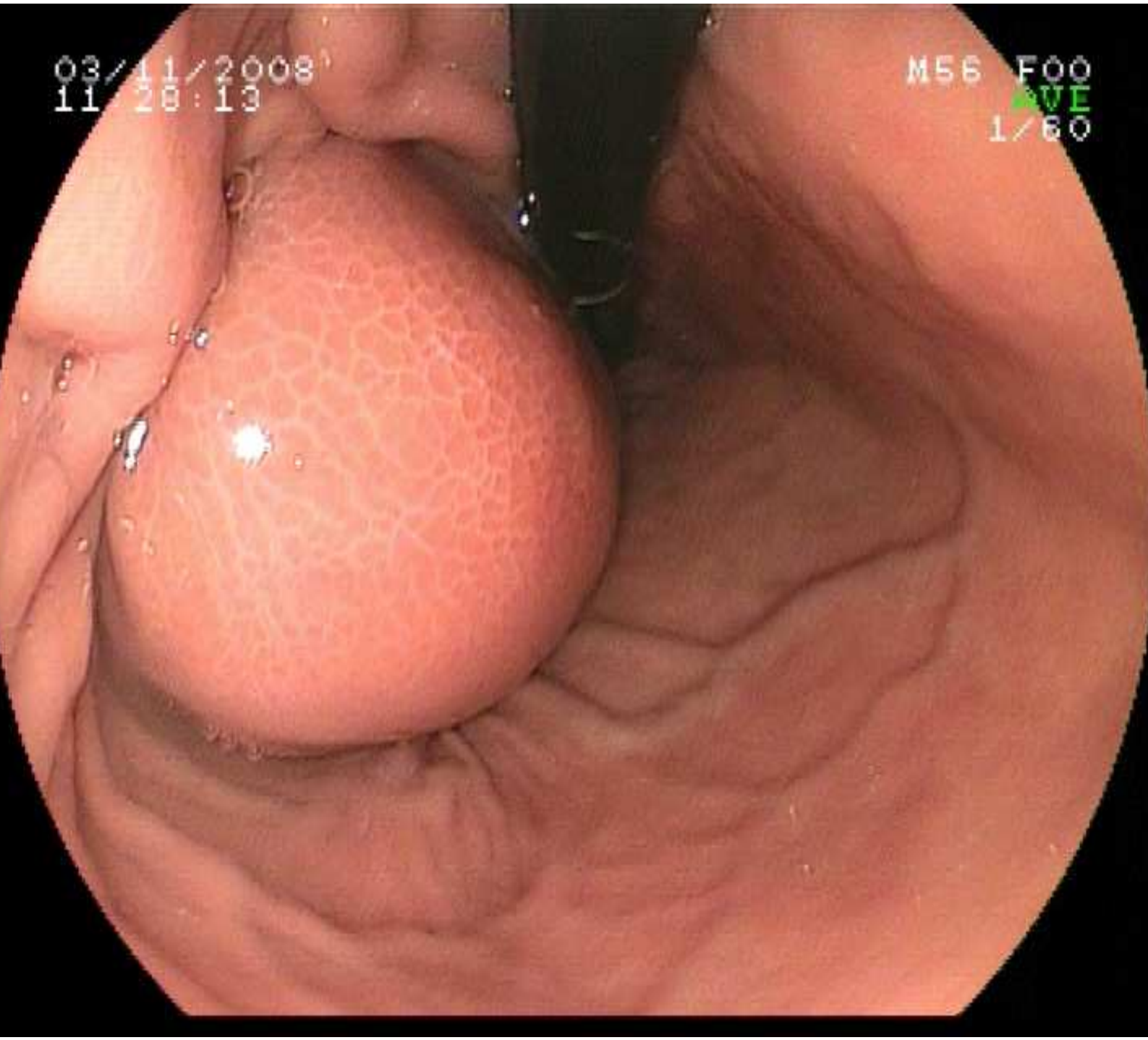
The shunting of esophageal collaterals or varices, are the most important clinically, because of their predilection to bleeding. The development of this portosystemic shunting depends upon a threshold portal pressure below which varices do not occur unless the portal pressure, as measured by the hepatic vein wedge pressure gradient is greater than 12mmHg, varices and variceal haemorrhage do not develop[39]. As all the four layers of veins in the wall of esophagus are intercommunicating, they all become engorged and elongated, dilated and tortuous when the pressure is above 12 mm of Hg. However, the deep intrinsic veins seem to bear the brunt of the insult and dilate massively becoming esophageal varices, which are seen endoscopically. These vessels lie in the lamina propria, where they are poorly supported by surrounding tissue bulge into the lumen.

Oesophageal varices are classified into,

- Grade 1: varices that looks small and straight.
- Grade 2: varices that are occupying less than 1 /3rd of the lumen
- Grade 3: varices that are large, coil shaped occupying > 1 /3rd of the lumen.
- Grade 4 : Near complete occlusion of oesophageal lumen.

GASTRIC VARICES

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Gastric Varices

Short gastric veins presenting at the fundus communicate with the deep intrinsic venous plexus of the esophagus, which due to back pressure changes result in gastric varices of fundal type. They are common in extrahepatic obstruction like splenic vein thrombosis. There is diffuse increase in arterio-venous communications between muscularis mucosa and dilated pre-capillaries. This is termed as congestive gastropathy[37]. They have a particular risk of bleeding and of damage eg. By Aspirin or NSAIDs. Therefore bleeding gastritis constitutes 30% of upper gastrointestinal tract bleeding in portal hypertension patients.

Gastric varices are classified primarily by their location as,

A. Gastroesophageal varices

Type I (GEV 1)- along the lesson curve (2-5cm in length)

Type II (GEV 2) – along the greater curve extending towards the gastric fundus

B. Isolated Gastric Varices

Type I (IGV 1) – Isolated cluster of varices in gastric fundus

Type II (IGV 2) – Isolated gastric varices in other parts of the stomach

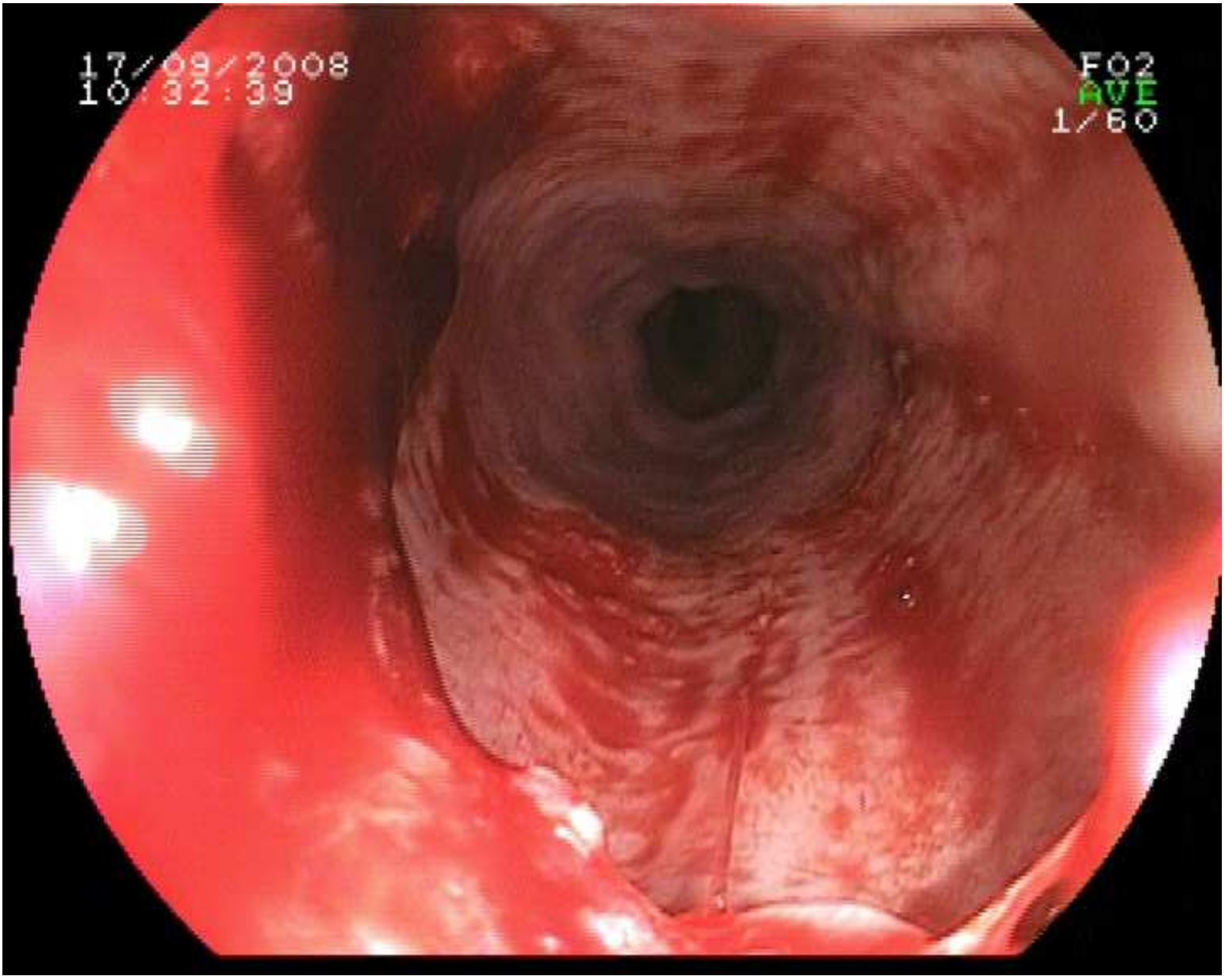
Upper Gastrointestinal tract tumours:

Oesophagogastric tumours are uncommon cause of acute upper gastrointestinal bleeding. The important benign type is gastrointestinal stromal cell tumour (GIST) arising from the muscle layers of the gastric or duodenal wall. Erosion through the mucosa gives a characteristic umbilicated in endoscopy. These tumours may cause major bleeding by eroding the underlying arteries. Acute GI bleeding due to malignant lesions are unusual (6%) arising from the oesophageal malignancies presents as massive bleeding due to aortic invasion. Significant gastrointestinal bleeding is uncommon with gastric cancer; however, hematemesis does occur in approximately 10% to 15% of patients[43].

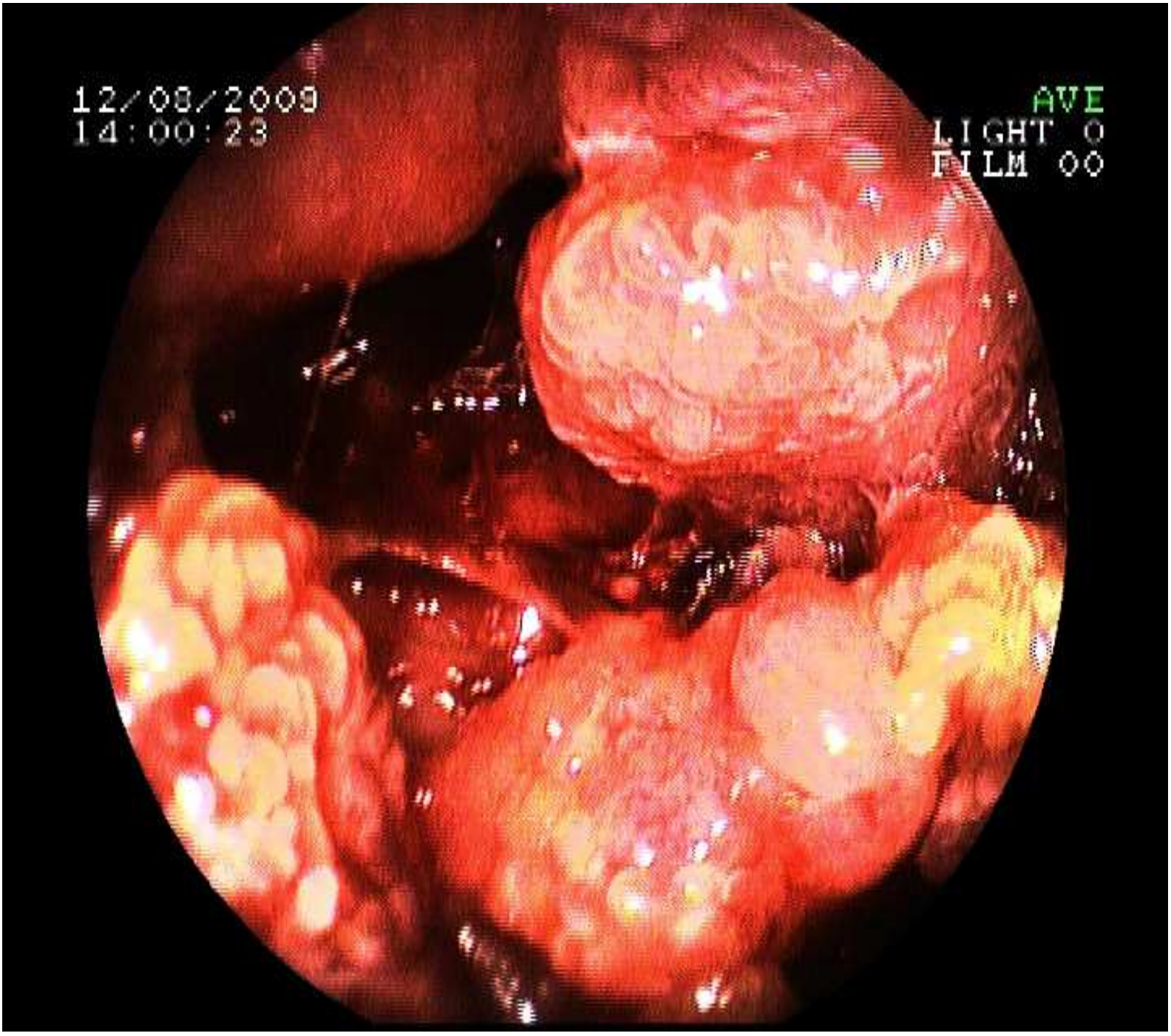
Dieulafoy lesion:

It is rare cause of Upper GI bleeding. It comprise about 3 – 6 % of all gastrointestinal bleeds in adults. They are thought to be of developmental malformation and are often called as, cirsoid aneurysm, and submucosal arterial malformation. They can occur anywhere in the GI tract, and most commonly in the proximal part of the stomach[40].

EROSIVE OESOPHAGITIS



ULCERO PROLIFERATIVE GROWTH



Arterio venous malformations:

Arteriovenous malformations (AVMs) are abnormal blood vessels seen in the gastrointestinal (GI) tract and are the source of bleeding. They are identified nowadays by the use of angiography[41]. Presentation could be of massive UGI bleeding or anaemia of chronic blood loss.

Other causes:

Other rare causes of UGIB should also be considered in patients with UGIB. For example, In patients with chronic pancreatitis who presents with UGIB, Hemosuccus pancreaticus must be excluded. Bleeding in these patients can be secondary to a pseudoaneurysm in peripancreatic blood vessels as a complication of pancreatic pseudocysts[42]. Hemobilia is another rare cause of UGIB that should be considered in the setting of recent hepatobiliary tree instrumentation, such as with ERCP (Endoscopic retrograde cholangio pancreatography) or laparoscopic cholecystectomy possibly due to the injuries of bile duct and hepatic artery. Aortoenteric fistula must be considered in patients with a history of intra abdominal vascular surgery, such as Abdominal aortic aneurysm repair. Post chemotheraphy or radiation sequelae may also be considered. Finally,

Iatrogenic injuries secondary to endoscopic procedures, such as percutaneous endoscopic gastrostomy tube placement, are also rare causes of UGIB. portal gastropathy, Ménétrier's disease, and watermelon stomach(Gastric Antral Vascular Ectasia) should also be considered.

Clinical features:

All patients presenting with Haemetemis or Malena with associated symptoms provides clue to the diagnosis as follows,

1. Symptoms due to Oesophagitis or Peptic ulcer disease.
2. Symptoms pertaining to Upper GI malignancies.
3. Symptoms consistent with the cause of portal hypertension
4. Symptoms of portal hypertension
5. Symptoms directed towards the esophageal Varices.

Clinical features vary depending upon the underlying causes like, Peptic ulcer disease usually have relationship of symptoms pertaining to food intake, Gastric malignancies have Ball rolling movements and symptoms suggestive of Gastric outlet obstruction[43] and cirrhosis patients have features of Liver failure like Spidernaevi, Foetor hepaticus, Gynaecomastia, Testicular atrophy, Palmar erythema, peripheral edema ,

Flapping Tremors and Hepatic encephalopathy [31]. Clinical features of portal hypertension like Ascites, Splenomeagaly with Hyper splenism, caput medusae should be noted. Patient may come with severe Haematemesis and Melaena in the case of ongoing or massive bleeding due to Erosive gastritis, Malignancies, Esophageal or Gastric varices.

Investigations:

Complete Blood Count:

*Haemoglobin and hematocrit , Total Count**,** Differential count **,** E.S.R, Packed Cell volume (P.C.V), Platelet count.*

Bleeding time, Clotting time, Pro thrombine Time

Renal Function Test:

*Blood Sugar**,** Blood Urea, Sr. Craetinine.*

Blood Grouping and Rh typing

Urine routine

Liver Function Test:

- Serum Bilirubin
- Serum proteins

- ❖ Albumin: Globulin ratio
- ❖ Serum Asparate amino transferase (AST, SGOT)
- ❖ Serum Alanine amino transferase (ALT, SGPT)
- ❖ Serum Alkaline phosphatise

Chest X ray PA view.

X ray Abdomen AP erect view.

ECG.

Barium Swallow and Barium meal: Linear filling defects in the distal esophagus and stomach can be made out depending upon the underlying pathology.

Endoscopy(Oesophago Gastro Duodenoscopy) : Gold standard for the diagnosis of varices . This is the single most important investigation when the patient comes with upper G.I. bleeding and Malena. The bleeding site and the underlying cause can be identified by the use of OGD. In Emergency situations it can be used diagnostic tool to identify the spurting artery, visible vessel / varices or stigmata of recent haemorrhage (SRH) and therapeutically to control bleeding[44].

Ultra sonography of Abdomen with Doppler evaluation

- ❖ Ascites
- ❖ Splenomegaly
- ❖ Hepato megaly
- ❖ Portal vein Caliber, splenic, hepatic vein and infra and intrahepatic IVC
- ❖ Angiography
- ❖ Measurement of portal pressure

Liver biopsy: To know the cause of intrahepatic portal hypertension.

MANAGEMENT:

Protocol for early management of acute upper gastrointestinal bleeding

Triage

Patients are prioritised depending upon the clinical presentation and decisions to be taken accordingly whether surgical intervention is needed or not.

Intensive care monitoring

Maintaining the circulation by accessing the central venous line, Nasal oxygen , urinary catheterisation to monitor the output, ryles tube insertion done to clear the blood filled stomach for endoscopy and also to asses the ongoing bleeding. Vital signs should be monitored periodically by using appropriate methods.

General supportive therapy

Endotracheal intubation should be attempted if needed[45]. Patients can be resuscitated by using intravenous fluid administration, compatible blood transfusion, cardio pulmonary resuscitation, and management of associated comorbid diseases, such as sepsis, liver disease or coronary artery disease[46]. OGD could be delayed until the patient is adequately resuscitated and stabilized with the available measures. Nasal oxygen must be given to counteract the blood loss which indirectly decreases the oxygen carrying capacity. Patients with acute UGIB must be kept nil oral for the purpose of the urgent need for OGD and for abdominal surgery if needed. They are also assessed for hypovolemic shock to determine requirements of intravenous fluid administration and blood transfusion, and attention should be paid for comorbid diseases, especially coronary artery disease.

Intravenous access is secured at two or more sites using 16 or 18 -gauge venflons.

Patients with active UGI bleeding should receive initial 500mL of crystalloids, during the first half an hour to maintain the blood pressure, while several units of packed redblood cells are cross matching and typed[46]. Fluid administration is subsequently raised if the blood pressure falls. Transfusion requirements are determined by many factors, including the age of the patient, presence or absence of comorbid ilnesses, cardiovascular status, hematocrit, and the quantity of blood loss, along with the current hematocrit level. Packed redblood cells are transfused in individuals who have significant blood loss, ongoing bleeding, and in those patients who manifest cardiac, renal, or cerebral ischemia. Patients who presents with variceal haemorrhage are conservatively transfused to a hematocrit of only 26 to 28 to avoid exacerbating the bleeding by increasing the portal pressure[47]. Fresh frozen plasma transfusion or Platelets transfusion is individualized according to multiple factors like, severity of bleeding, bleeding rate, presence of other bleeding diathesis, qualitative platelet defects, induced by NSAIDs [48].

ROLE OF ENDOSCOPY:

OGD stands as the prime diagnostic and therapeutic tool for UGIB[49] . It accurately identifies the bleeding site and determines the specific cause and in more than 90% of individuals with acute UGIB [50]. OGD is the principal modality in diagnosing the type of bleeding and therapeutically it reduces the surgical intervention. Befor OGD, it advisable to provide proton pump inhibitor therapy[51]. Urgent OGD for UGIB is ideal, which significantly improves the clinical outcome in certain conditions like variceal bleeding and severe ongoing bleeding[52]. Stigmata of recent haemorrhage could be identified by performing OGD as early as possible. From this, the site and number of lesions are identified. Multiple scoring systems like Rockall, forrest classification etc are used for prognostic purposes and triage of patients with UGIB[53]. Local adrenaline injection, electrocautery or argon plasma coagulation(APC), and mechanical therapies like endoclips or banding are the available therapies with endoscopy. Combined with the clinical presentation and OGD findings, risk stratification can be made[54].

PEPTIC ULCER DISEASE:

These ulcers are seen as craters in endoscopy. For controlling the bleding, adrenaline injection is the agent of choice, which should be given

at the site of bleeding or surrounding the ulcer. Sclerosants like ethanol, sodium tetradecyl sulphate can also be useful [55]. The use of electrocautery, argon plasma coagulation was also justified.

Reflux esophagitis:

The endoscopic findings are erythematous mucosa, edema with exudates, ulceration and bleeding with increased vascularity. Ulcers due to oesophagitis can be efficiently treated with adrenaline injection or by using ablation therapies[56].

Mallory- weiss tear:

A linear and longitudinal tear that are seen over the osophago gastric junction. Sometimes an erosion or scab are also identified during endoscopy. The role of therapeutic endoscopy is under evaluation[57].

Cameron lesion:

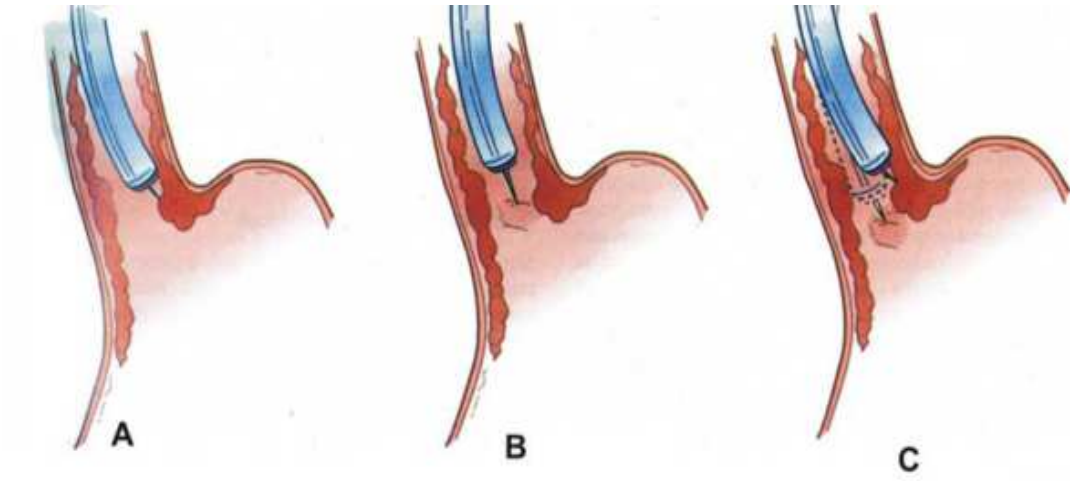
The ulcers or erosions, that are seen over the gastric part within the hiatus hernia are termed as Cameron lesions. They are asymptomatic and are incidentally diagnosed during endoscopy. These lesions are uncommon cause of acute bleeding[58] and the therapeutic modalities are injection with adrenaline or APC.

Portal gastropathy:

This condition looks like an intense red lesion in a mosaic background, commonly noted in the fundus. Portal hypertension is commonly associated with it. Due to the diffuse nature of the lesion, therapeutic modalities in endoscopy are not useful[59].

Oesophageal and Gastric varices:

Endoscopic sclerotherapy:



Techniques of endoscopic sclerotherapy. A flexible endoscope is used for intravariceal injection (A), paravariceal (submucosal) injection (B), and combined paravariceal and intravariceal injection (C).

Obliteration of esophageal varices by injecting sclerosants directly into the channel (Intravariceal), beside the channel (Paravariceal) or combination of both is called sclerotherapy[60].

Indications:

1) Emergency sclerotherapy can be performed immediately at the time of the diagnostic endoscopy.

2) It can be delayed until after the variceal haemorrhage has been controlled by conservative measures.

3) In patients for whom no other treatment is available, including those in whom surgery carries high risk, those who have undergone surgery but continue to bleed.

4) In patients prior to definitive surgery so that the patients condition can be improved. Hence reduces the mortality.

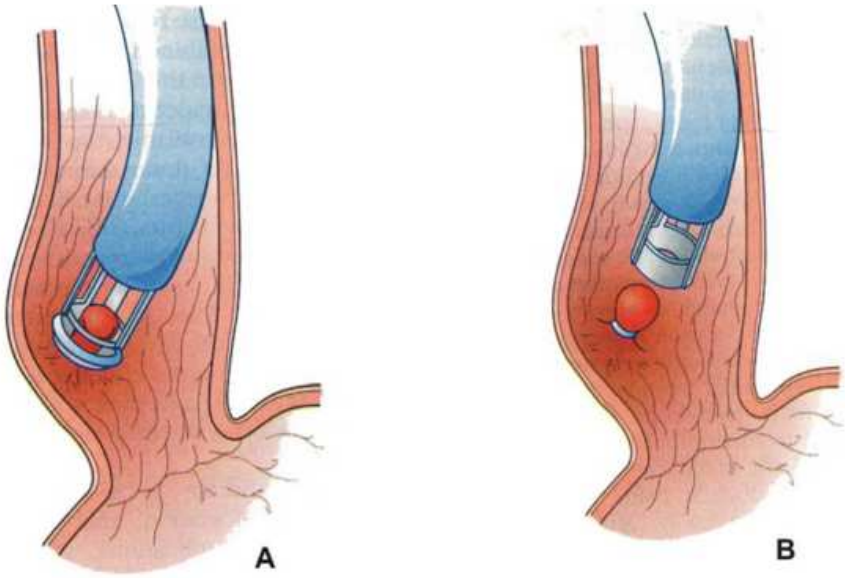
Mechanism of action:

Intra variceal sclerosant injection acts by causing thrombosis (damage the intima) thus stopping the bleeding and preventing rebleed from particular vein. In paravariceal injection sclerotherapy bleeding stops by two mechanisms:

1) By external compression of the bleeding varices (Peri vascular fibrosis)

2) By variceal contraction.

Endoscopic variceal ligation:



Indications:

1. In acute variceal bleeding, the band ligation has to be done immediately after the diagnosis, to control the bleeding.
2. In the long term eradication of the esophageal varices.
3. Prophylactic band ligation of the esophagal varices has also been reported
4. In high-risk patients the band ligation is indicated, till the patients recover from the risks and become fit for surgery.
5. Indicated in patients who are not fit for surgery (definitive surgery).
6. Indicated in the esophageal variceal eradication along with the low volume sclerotherapy (intravariceal)[61]

Mechanism of action:

Band ligation of esophageal varices acts by mechanical obstruction, leads to strangulation of variceal tissue. The strangulation of the varices ligated is followed by ischaemic necrosis of the mucosa and sub mucosa of the varix. At 3-7 days after treatment, sloughing of the ligated tissue, at the site with shallow ulcerations occurs (1-2 mm deep) and they are 6-10 mm in diameter. At 14-21 days minimal residual varices are present, the vascular structures in the submucosa will be replaced by matured scar tissue. After 50-60 days at variceal site mature scar tissue, without any stricture is seen.

Benign and malignant Upper gastrointestinal tumors :

These are all rare causes of UGIB. Benign conditions like GIST and leiomyomas are seen as extraneous compression in endoscopy. MALTomas are seen as polypoidal mass with cerebroid like mucosal folds. Malignancies, more commonly adenocarcinoma stomach are seen as an ulcerative growth, ulcero proliferative lesion, bleeding irregular mass or stricture. Linitis plastica, seen as a noncompliant stomach wall. Secondaries in the stomach are visualised as polypoidal erosions or mass. They tends to rebleed and are having worst prognosis[62].

Dieulafoy lesion:

During endoscopy, these lesions are visible as an elevated lesion with erosions around it. In majority of cases, dielaufoy lesions are seen in the upper half of the stomach along the lesser curvature. It is about 3 – 6 mm in diameter. Adrenaline injection, argon plasma coagulation, ligation with bands or clips are useful to arrest bleeding[63].

Angiodysplasia:

These lesions appear as dark red, thick network of vessels, 2 – 6 mm in diameter. Initially, adrenaline injection is used to control haemostasis followed by APC[64].

Gastric antral vascular ectasia:

The lesions which are seen as multiple folds radiating from the pylorus upto the antral region with red streaks at the proximal ends. They also termed as watermelon stomach. Due to its superficial and diffuse nature of the lesion, thermal ablation are useful[65].

Aortoenteric fistula:

It carries a very high mortlity as it necessitates the emergency endoscopy. Aortoenteric fistula commonly seen in the lower 3rd of the duodenum[66].

Sometimes, a prosthetic mesh can also be seen during endoscopy. OGD must be deferred after identifying such lesion, as therapeutic procedures may cause alarming haemorrhage on disturbing the lesion.

Repeat esophagogastroduodenoscopy

Repeat endoscopy is useful at times to identiy the missed out lesions. Rebleeding from the lesions occurs with in three days of the first endoscopy and the relook endoscopy is generally not advisable regularly[67].

RISK STRATIFICATION:

Several scoring systems have been developed to help predict the outcome of patients and to improve patient management and promote cost-effective use of hospital resources .

The Rockall scoring system is used for risk stratification with the use of clinical presentation, comorbid factors and endoscopy findings. It can be done befor and after endoscopy for accurate calculation. Mortality can be predicted with the use of it.

A total Rockall score of < 3 is predictive of low risk of adverse outcomes and is appropriate for early discharge and/or outpatient management while

a score of > 8 is predictive of high mortality[68]. With Rockall scoring system, individuals with high and low risk strata are calculated.

ROCKALL RISK SCORING SYSTEM

Rockall Risk scoring system	
Variable	Scores
Age (Years)	
< 60 Years 60 - 79 Years > 80 Years	0 1 2
Shock	
Pulse < 100/min, SBP>100 mmHg Pulse > 100/min, SBP>100 mmHg Pulse < 100/min, SBP<100 mmHg	0 1 2
Co-Morbid Conditions	
No Major Co-Morbidity Cardiac Failure, Ischemic Heart Disease Renal Failure, Liver Failure, Disseminated Malignancies	0 2 3
Diagnosis	
Mallory Weiss tear, No Lesion Identified All other Diagnosis Malignancy of Upper GI Tract	0 1 2
Major Stigmata of Recent Haemorrhage	
None / Dark Spot Only Blood in Upper GI Track, Adherent clot, Visible or Spurting Vessel	0 2

Evaluation of the bleeding lesion was determined in **Forrest classification** by using OGD findings alone and does not include clinical parameters[69].

Blatchford risk scoring system was used in patients with UGIB to predict the clinical outcome , using only the clinical parameters without endoscopic evaluation of bleeding lesion.

CONSERVATIVE MANAGEMENT:

Management of Non-variceal bleeding:

Gastric acid inhibits platelet aggregation, impairs clot formation, and promotes fibrinolysis; therefore, inhibiting gastric acid and raising the intra gastric pH to 6 or more may promote clot formation and decrease the risk of rebleeding.

High-dose PPI therapy is defined as an initial bolus (Pantoprazole 80 mg) followed by continuous infusion (Pantoprazole 8 mg/h) for up to 72 h. Omeprazole can also be used with best results.

Since H.Pylori is the leading causative factor in Peptic ulcer disease, the following regimen should be considered[70].

Anti-H.Pylori regimen:

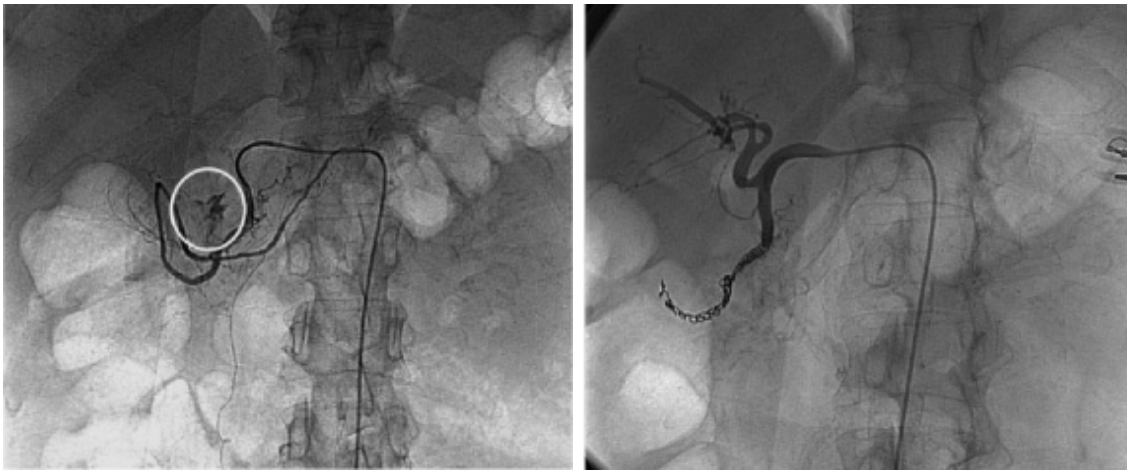
PROTON PUMP INHIBITORS THERAPY bd + CLARITHROMYCIN 500mg bd + AMOXYCILLIN 1g bd	10 – 14 days
PROTON PUMP INHIBITORS bd + CLARITHROMYCIN 500mg bd + METRONIDAZOLE 500 mg bd	10 – 14 days
PROTON PUMP INHIBITORS bd + BISMUTH SUBSALICYLTE 525mg qid + METRONIDAZOLE 250mg qid + TETRACYCLINE 500mg qid .	10 – 14 days

Radiological approach

Transcatheter coil embolisation is commonly used as a therapeutic approach in patients with unidentifiable and uncontrollable bleeding with the routine measures Bowel ischaemia is less as there is good collateral supply of the stomach and duodenum[71].

Safety and effectiveness of the procedure was explained in recent studies, in patients with acute GI bleeding.

BEFORE AND AFTER EMBOLISATION OF GASTRODUODENAL. A.



Partial splenic artery embolization (PSE) has been performed to treat Oesophageal and Gastric varices including portal hypertensive gastropathy.

Management of Variceal bleeding:

Numerous agents have been studied for the prevention and treatment of variceal hemorrhage. In practice, the list consists of vasopressin and its analogues, somatostatin and octreotide, nonselective β- blockers and nitrovasodilators.

I. Vasopressin and its analogues:

Vasopressin causes splanchnic arteriolar vasoconstriction and decreases portal tributary inflow with a resultant decline in portal pressures Standard dose: Vasopressin 20 units bolus over 20 minutes and continuous IV

infusion 0.2-0.4 unit/min with NTG 40 μg/min. The morbidity of vasopressin has led to the development of analogues with fewer side effects such as terlipressin. This drug does not increase plasminogen activator activity but has the same effects as vasopressin on the coronary vasculature.

II. Somatostatin and its analogues

It is a naturally occurring tetradecapeptide found in the GIT. Octreotide is a structural analogue of somatostatin. Somatostatin is administered as a 250 μg /hour IV bolus followed by continuous infusion of 250μg/hour for 2-4 days.

Octreotide is given as 50μg IV bolus followed by infusion of 25-50 μg /hour. Because somatostatin and octreotide have to be administered parenterally, their role has primarily been restricted to the management of the acutely bleeding cirrhotic patient[72].

III. β- adrenergic Antagonists

They cause splanchnic arteriolar vasoconstriction and decrease portal venous inflow. Propranolol is the prototype nonselective β-blocker.

In the long term, propranolol maintains a portal hypotensive effect in most subjects, but tachyphylaxis occurs in 50% to 70% of patients.. β-blockade

is associated with numerous side effects such as bronchoconstriction, heart failure and impotence in cirrhotics. Their administration does not impair the hemodynamic response to acute blood loss.

IV. Nitrovasodilators:

Nitric oxide is one of the most potent vasodilators and plays an important role in pathophysiology of portal hypertension. Patients treated with isosorbide mononitrate together with either propranolol or nadolol had a greater sustained portal hypotensive effect.

Balloon Tamponade:

They are highly effective in controlling esophageal variceal bleeding, but temporarily. There are three types of tubes available

They are:

a) Linton-Nachlas tube: It has only gastric balloon and three lumen.

b) Sengstaken Blakemore tube: Both gastric and esophageal balloons are present and have three lumen.

c) Modified sengstaken Blakemore tube or Minnesota tube:

It has four lumen, one excess to prevent aspiration pneumonia. After testing the balloons for leak, it should be passed under sedation or anaesthesia (Local or General) in operation theatre, through nostril. Once the tube in the stomach is confirmed by aspiration and easy distensibility of the gastric balloon, (300-400 ml of air is used for inflation). So that the pressure is maintained between 20-30 mm of Hg. Tube is secured to the forehead with slight traction. Inflate the esophageal balloon to a pressure of 40 mm Hg.

Removal of the tube:

24 hrs after inflating the esophageal balloon if there is no bleeding, deflate the bulbs and remove the tube. If bleeding is present, we can keep the tube for further 24 hrs.

Results:

Several studies have shown 60-70% effectiveness. However, the rebleed rate is as high as 40-60%. Hence, this is as effective as pharmacological control.

Transjugular intrahepatic portosystemic shunts

In this procedure, a communication is made inbetween the portal vein (intrahepatic branch) and hepatic vein. Haemostasis is achieved > 80% of

cases. Model of End Stage Liver Disease is the good mortality predictor after the procedure. It is not useful in patients with multi organ failure. CCF, pulmonary hypertension and portal vein thrombosis are contraindications of TIPSS[73].

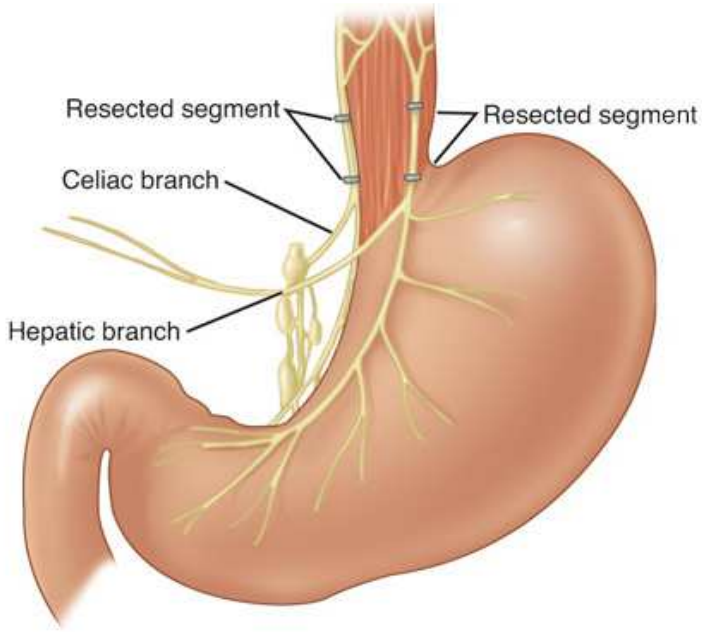
SURGICAL MANAGEMENT:

Non varicel bleeding:

Despite major advances in endoscopic treatment, the incidence of emergency surgery has not significantly changed. Today, most patients undergoing operation for bleeding peptic ulcer have simple oversewing of a bleeding ulcer, or simple patch of a perforated ulcer, Truncal vagotomy or distal gastrectomy. Surgical interventions, such as ligation of the bleeding vessel or excision of the aneurysm, should be considered if embolization fails or is contraindicated in case of Hemosuccus pancreaticus.

Vagotomy:

These surgical procedures are associated with ulcer recurrence as carried out in emergency situations. Vagotomy with drainage procedures carry minimum ulcer recurrence rate [74].



Gastrectomy:

Surgical resection appears to be the only curative treatment for gastric cancer and most patients with clinically resectable loco regional disease should have gastric resection. The standard operation for gastric cancer is radical subtotal gastrectomy. Reconstruction is usually by Billroth II gastro jejunostomy, but if a small gastric remnant is left (<20%), a Roux-en-Y reconstruction is considered. The operative mortality is around 2 to 5%.

Variceal bleeding:

1. **Portosystemic shunting**: They are aimed at lowering the portal pressure and diverting the portal flow from around the gastro-esophageal area.

A**) Total shunts**:

1) End to side portacaval shunt

2) Side-to-side portacaval shunt

3) Mesocaval shunt

4) Proximal splenorenal shunt

B) **Selective shunts**:

1) Distal splenorenal shunt

2) Coronary caval shunt

C**) Partial shunts**

Small diameter H-graft (sarfeh shunt)

This is the standard for both the emergency and elective treatment. End to side Porta caval shunt was introduced in 1940's, itself, the operative mortality is much higher in emergency situation when used as a last resort was 50% mortality rate, in elective patients it may be as low as 19% in best hands[75]. There is no role for prophylaxis.

2**. Devascularization procedures**:

a. Splenectomy

b. Esophageal transection

c. Esophageal transection and devascularization (Suguira procedure)20

Many shunt techniques evolved of these distal splenorenal shunt is said to be most advantageous.

The devascularization procedures carry mortality rate of 33% as compared to endoscopic procedures which is 24%.

Contra-indications for emergency surgery are,

1. Presence of acute alcoholic hepatitis.
2. Marked coagulopathy that is uncorrectable
3. Presence of major systemic complications related indirectly to liver disease such as acute renal failure, frank sepsis etc.

Child class ‘c’ is not a contraindication, infact they are resistant to conservative treatment.

Liver Transplantation: Theoretically, it is the ideal therapy for all patients with chronic liver disease complicated by portal hypertension and variceal haemorrhage. This re-establishes low resistance portal outflow through the liver. Considering the availability of the donor, cost of both the procedures and immunosuppressive medication it is indicated for the treatment of selected patients with end stage liver disease that is medically and surgically intractable.

MATERIALS AND METHODS

STUDY DESIGN **:** Prospective study.

SAMPLE SIZE **:** 100 patients admitted with upper GI bleeding .

PLACE OF STUDY **:** Trauma ward, Department of General Surgery, Government Stanley Medical College Hospital, Chennai.

PERIOD OF STUDY **:** 1 Year.

INCLUSION CRITERIA:

All patients with age groups 20 to 85 years admitted with Upper GI bleeding in Trauma ward.

EXCLUSION CRITERIA:

Children and patients with age groups below 20 years with Upper GI bleeding.

METHODS:

- A Proforma will be made that includes detailed history, physical examination , basic investigations and other relevant investigations required.

- Clinical diagnosis will be made accordingly.

- Risk stratification will be done using Rockall risk scoring system.

- Triage, intensive monitoring and general supportive therapy done for the patients will be recorded.

- Endoscopic findings and various modalities of treatment are compared and analysed.

RESULTS

Total number of cases – 100.

Mean Age of presentation – 45 years.

Male : Female ratio – 89 : 11

CHART - 1

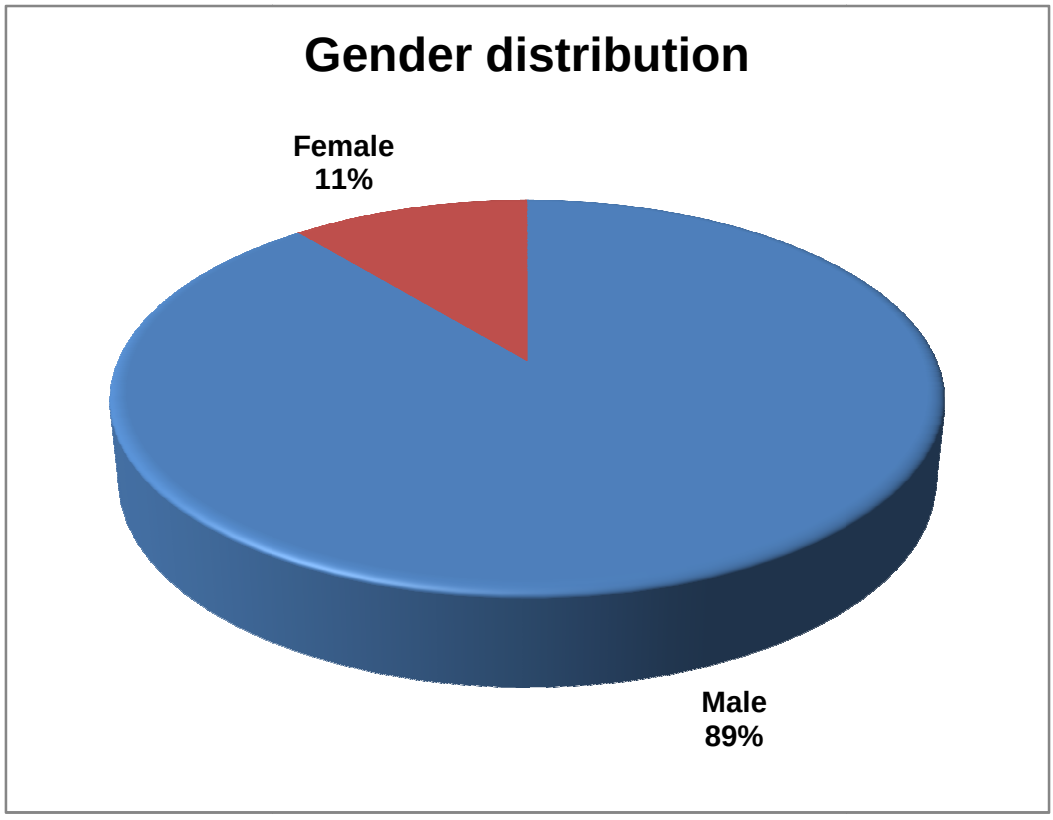
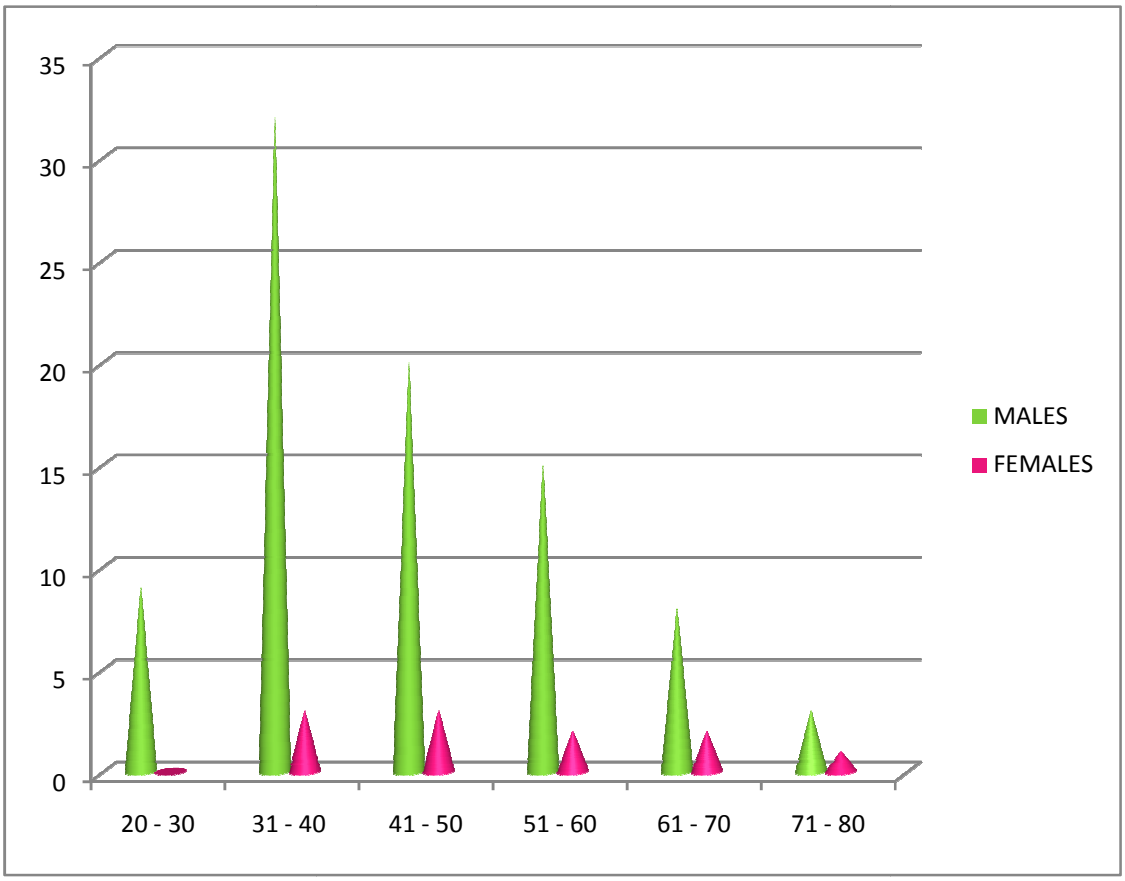


CHART – 2

Age distribution among males and females



Clinical severity of Upper Gastrointestinal bleeding:

TABLE - 1

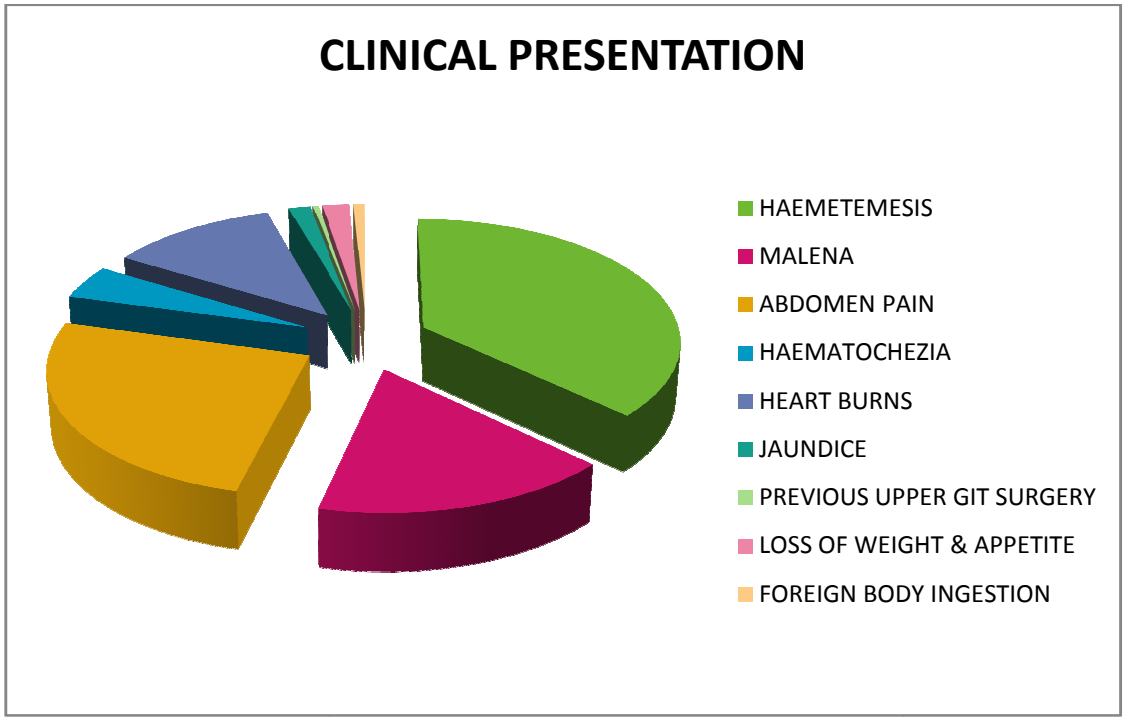
Mild (< 500 ml)	58
Moderate (500 – 1500 ml)	36
Severe (> 1500 ml)	6

Clinical presentation :

TABLE.2

Haemetemis	100%
Malena	45%
Abdomen pain	69%
Haematochezia	12%
Heart burns	32%
Jaundice	04%
Previous Upper GIT surgery	01%
Loss of weight and appetite	05%
Foreign body ingestion	02%

CHART - 3



Co-morbidities:

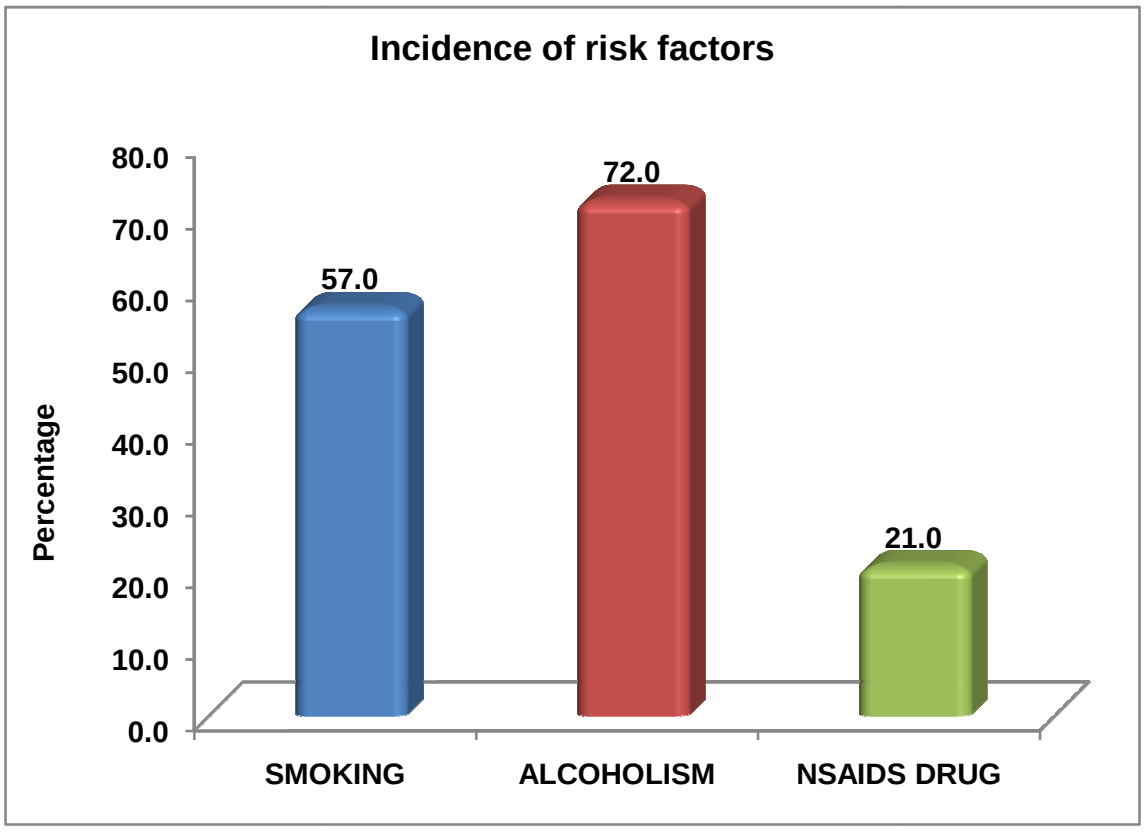
TABLE.3

Hypertension	12%
Diabetes	11%
Coronary Artery Disease	19%
Chronic liver disease	05%

Role of Risk factors :

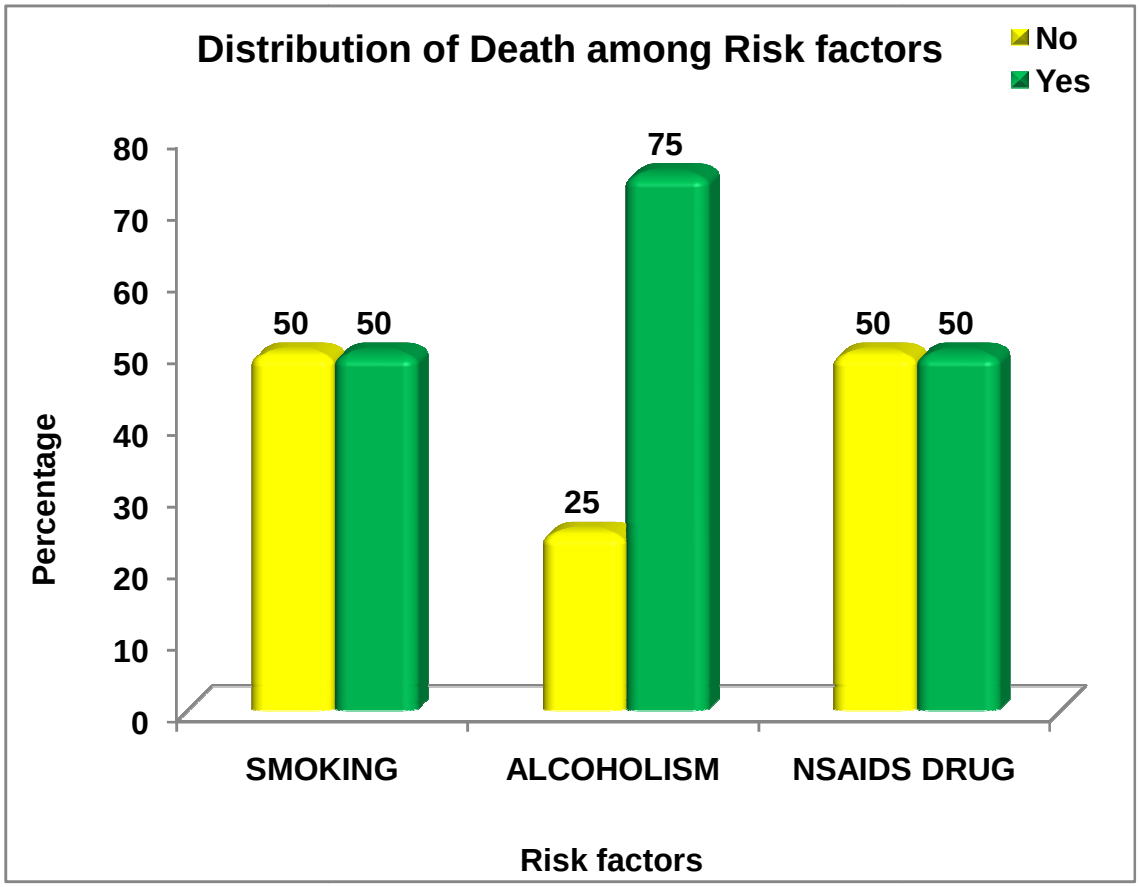
Smoking , Alcoholism and NSAIDS drug intake appears to be the major risk factors in patients presenting with Upper GIT bleeding.

CHART - 4



Alcoholism is one of the major risk factor associated with Upper Gastrointestinal bleeding in our study.

CHART - 5



Alcoholism tends to be the major risk factor causing mortality in Upper Gastrointestinal bleeding in our study.

OGD findings in our study :

TABLE – 4

OGD FINDINGS	**Frequency**	**Percent**
GASTRITIS	25	25
EROSIVE GASTRITIS	13	13
OESOPHAGITIS	9	9
DUODENITIS	8	8
MALLORY WEISS TEAR	5	5
NORMAL STUDY	4	4
DUODENAL ULCER	4	4
GASTRITIS &DUODENITIS	3	3
DUODENAL ULCER WITH BLEED	2	2
DUODENAL ULCER WITH CLOT	2	2
EROSIVE GASTRITIS & LAX LES	2	2
GASTRIC ULCER	2	2
LESSER CURVATURE GROWTH	2	2
OESOPHAGEALVARICES	2	2
OESOPHAGITIS& GASTRITIS	2	2
ANTRO PYLORIC GROWTH	1	1
BLEEDING FROM AMPULLA	1	1
DIEULAFOY LESION	1	1
DUODENAL ULCER & LAX LES	1	1

FOREIGN BODY - NEEDLE	1	1
FUNDAL VARICES&DUODENITIS	1	1
GASTRIC POLYP	1	1
GASTRIC ULCER WITH CLOT	1	1
GASTRIC ULCER WITH SLOUGH	1	1
GASTRIC VARICES	1	1
LOWER OESOPHAGEALVARICES	1	1
OESOPHAGEAL EROSION	1	1
OESOPHAGITIS&GERD	1	1
OESOPHAGITIS& LAX LES	1	1
STOMAL ULCER	1	1
Total	**100**	**100**

CHART - 6

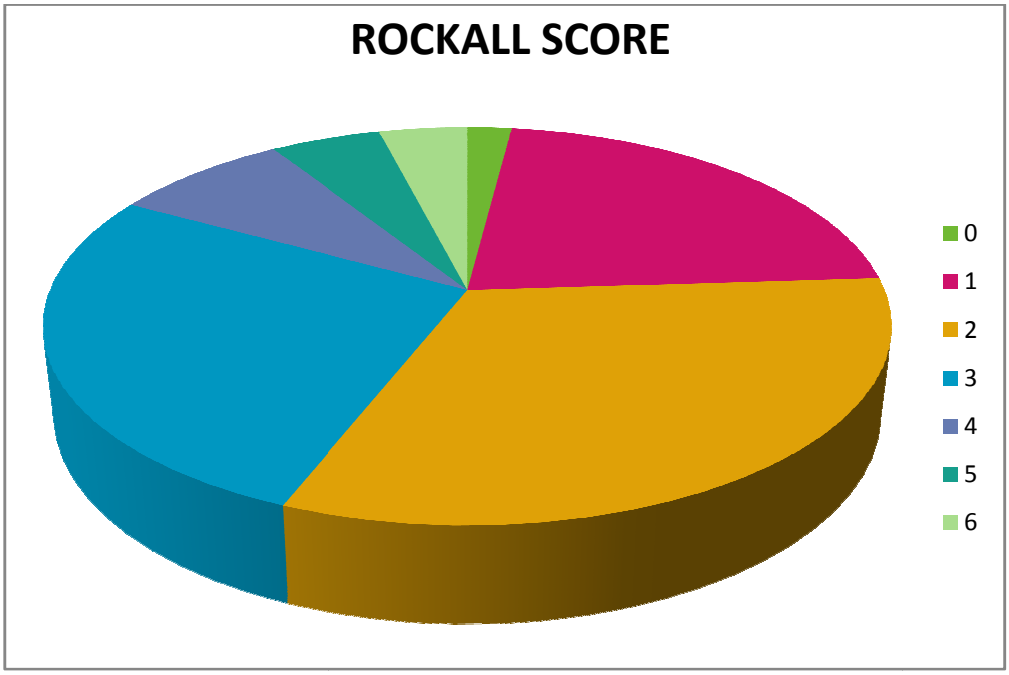


TABLE – 5

ROCKALL SCORE	Frequency	Percent
0	2	2.0
1	22	22.0
2	32	32.0
3	27	27.0
4	8	8.0
5	5	5.0
6	4	4.0
Total	**100**	**100.0**

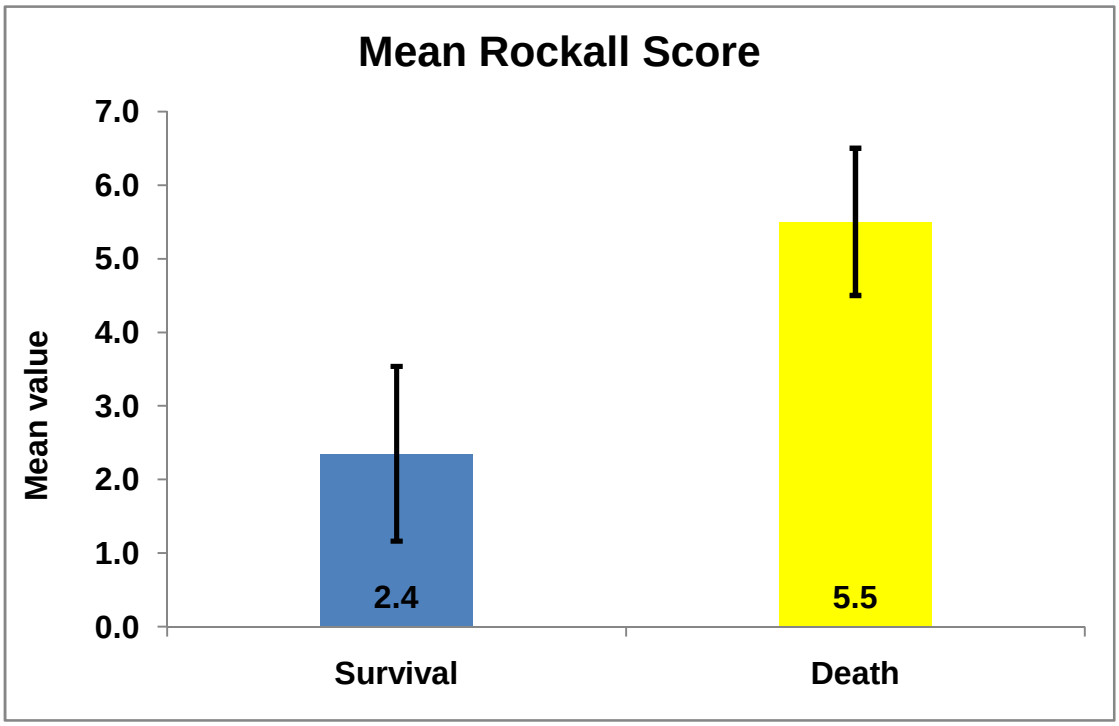
Impact of Rockalls score with mortality:

Independent samples t-Test to compare mean rockall score between Survival and Death

TABLE – 6

	DEATH	N	Mean	Std. Dev	t-Value	P-Value
ROCKALL SCORE	Survival	96	2.35	1.187	5.215	<0.001
	Death	4	5.50	1.000		

CHART - 7



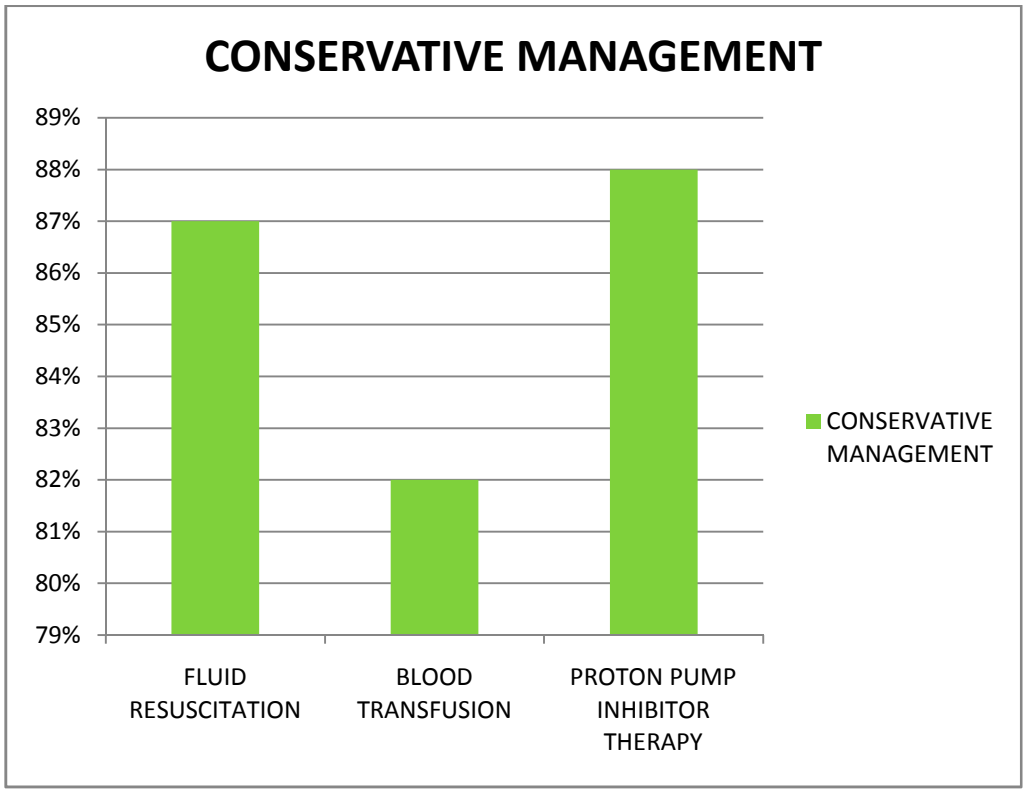
Conservative management :

Initial Fluid Resuscitation (crystalloids & colloids) - 87%

Blood transfusion - 82%

Proton pump inhibitor therapy - 88%

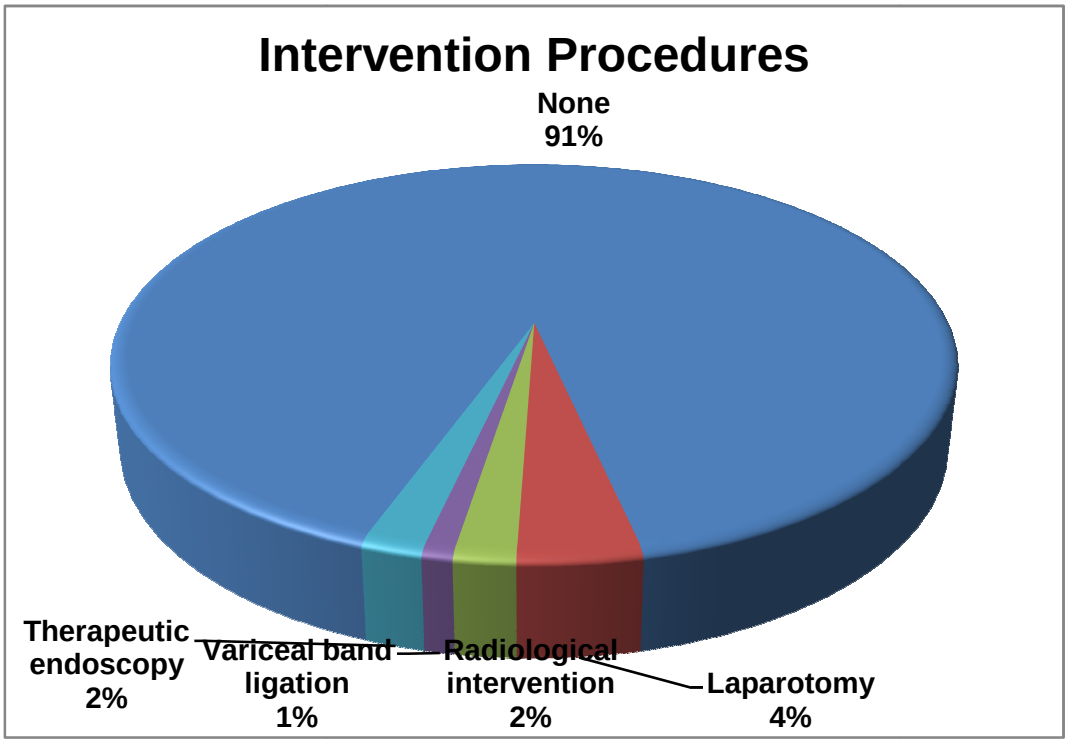
CHART.8



Initial fluid resuscitation with crystalloids and colloids were given in 87% of patients, 82% of patients were given blood transfusion and 88% of patients were managed with proton pump inhibitor therapy.

Intervention procedures:

CHART - 9



* **Therapeutic endoscopy** : - Gastric polypectomy , Foreign body removal and variceal band ligation.

* **Radiological intervention** procedures : Coil embolisation.

* **Laparotomy** :

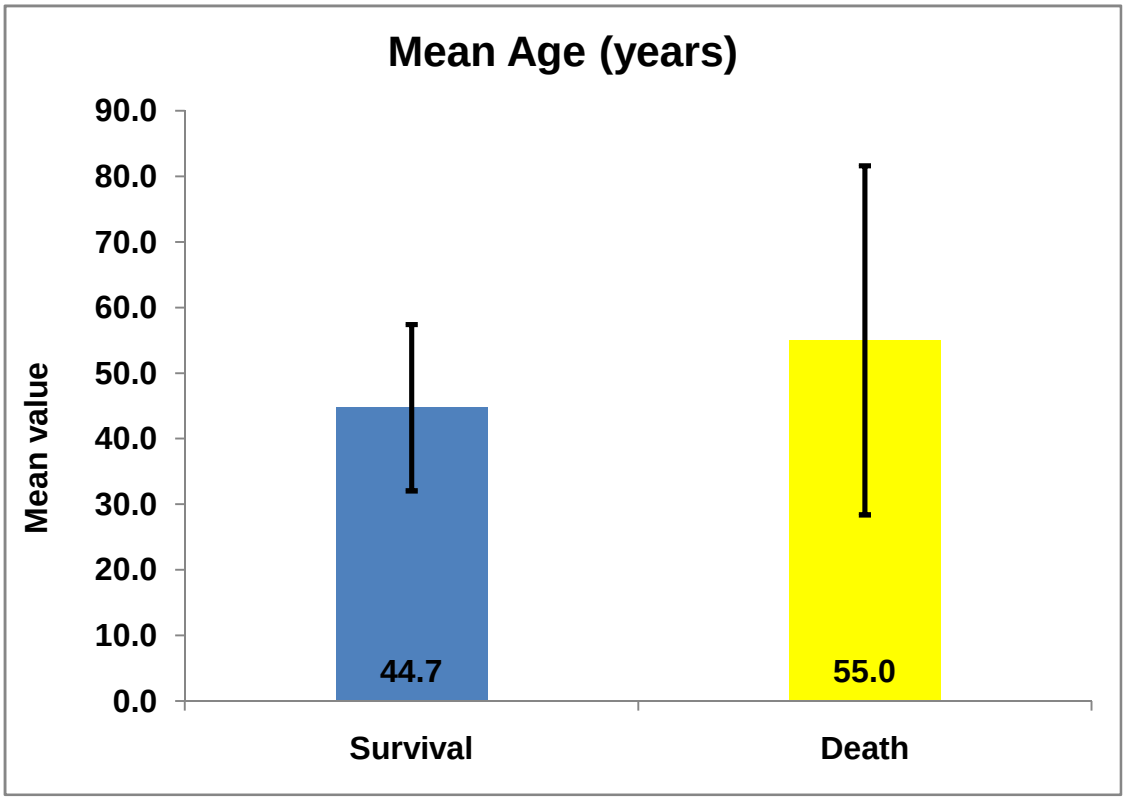
  - Under running suture over the bleeding vessel with Truncal vagotomy and pyloroplasty.
  - Subtotal Gastrectomy with Roux-en-Y anastomosis and D 2 resection.
  - Palliative Gastrojejunostomy.
  - Feeding Jejunostomy.

TABLE - 7

Independent samples t-Test to compare mean age between Survival and Death in Upper GI bleeding.

	DEATH	N	Mean	Std. Dev	t-Value	P-Value
AGE	No	96	44.72	12.677	0.769	0.497
	Yes	4	55.00	26.608		

CHART – 10 : MORTALITY IN UPPER GI BLEEDING



DISCUSSION

Upper Gastrointestinal bleeding is one of the most common emergencies in the surgical department that carries 6 to 10 % mortality worldwide[1]. Our study was a prospective clinical study on evaluation and management of Upper gastrointestinal bleding in patients admitted in our institute (Government Stanley Medical College Hospital) in one year. We excluded children and patients under the age of 20 years from this study. We assessed the clinical presentation, co-morbidities, associated risk factors, OGD findings (within 24 hours of presentation), need of initial resuscitation, blood transfusion and intervention procedures like therapeutic endoscopy, radiological intervention or emergency laparotomy.

A total of 100 cases were take up for the study. In these patients, the mean age of presentation was 45 years with a standard deviation of 13. Minimum and maximum age of presentation in the final study was 20 and 79 years respectively with sex distribution predominantly seen among males (89%) than females (11%). The maximum number of age group is between 31 – 40 years as shown in the chart.2.

The severity of bleeding was classified into mild, moderate and severe depending upon the quantity of blood loss. Majority of patients (58%) were presented with mild haemetemesis (< 500 ml), 36% 0f

patients presented with moderate haemetemesis (500 – 1500 ml) and only 6% presented with severe haemetemesis (> 1500 ml) as shown in Table.1. We had 58 patients in mild, 36 patients in moderate and 6 patients in severe form of which 4 patients were died inspite of resuscitative measures.

All of our patients admitted with haemetemis had Abdomen pain in 69 of them. About 45 patients had malena, 12 patients were presented with haematochezia and 32 patients had history of heart burns. History of anorexia and weight loss were observed in 5 patients while jaundice were noted in 4 patients as shown in Table.2 and Chart.3.

The mortality rate was 4% in our study with mean age of survival and mortality being 44 and 55 year respectively. Thus elderly people withstand less well to UGIB than younger patients. The major contributing factor that leads to death was blood loss, which inturn leading to hypovolemic shock.

The incidence of risk factors like Smoking, Alcoholism and NSAIDs drug intake were considered in our study and the impact with mortality have also been been compared. Smoking is seen in 57 % of patients while the incidence of alcohol was seen with 72 %. Around 21 % of patients were having NSAIDs intake. This was surprisingly a small number compared to other studies like Theocharis GJ, Thomopoulos KC et al [21].Most of the

patients took NSAIDS for arthritis and myalgia. Thus, it can be clearly delineated from the Chart.4 that alcoholism is one of the major risk factors causing Upper GI bleeding and almost, always associated with Peptic ulcer disease. Mortality rate also stands high in patients with alcoholism (75%) than the other two risk factors (50%) as shown in Chart.5.

The associated co-morbid illness in our study were Hypertension (12%), Diabetes (11%), Coronary Artery Disease (19%) and Chronic liver disease (5%) as noted in Table.3. One of our patients died had associated history of old coronary artery disease and another patient had chronic liver disease leading to portal hypertension. Thus, it can be stated that, associated co-morbid factors had a strong link with mortality.

OGD is very useful to delineate the site of bleeding and to facilitate targeted therapy. It defines the low risk and high risk strata and helps to identify the appropriate candidates for a period of hospital stay or intervention procedures if neded. Risk – stratification scores that incorporate endoscopic data, such as the complete Rockall score, propose that such scores are superior to clinically based scores because they define a greater proportion of all patients at low risk for adverse outcomes related to acute upper gastrointestinal bleeding. The pattern of Upper GI bleeding in our study was shown in Table.4. The commonest OGD finding in our

study was Gastritis (25%), followed by Erosive gastritis (13%). Thus, around 38% of the patients had gastric inflammation or erosions, of which 32 patients were alcoholics. The second commonest cause was found to be Oesophagitis (9%), followed by Duodenitis (8%) and Mallory weiss tear (5%). There was not able to identify any lesion in about 4% of the individuals. More than a single site lesion like Gastritis + Duodenitis, Oesophagitis + Gastritis can be seen in 3 and 2 % of patients respectively while Gastric and Duodenal ulcers were seen in 2 and 4% respectively. Stigmata of recent haemorrhage (SRH) were found in 5 patients with UGIB. Malignancies were noted in 4 patients of UGIB. In our study, stomal ulcer bleeding was seen only in 1 patient. A foreign body, needle was the cause of haemetemesis in one patient. Variceal bleeding from Oesophageal and fundal varices constitute 4% of UGIB in our study. According to the study by Boonpongmanee et al[27], the most common endoscopic findings were Gastric ulcer (23.1) and oesophageal varices (23.1), followed by Duodenal ulcer (13.9), Mallory weiss tear (10.2), Gastritis (4.7), Duodenitis (3.7) and Oesophagitis (3.7). According to the study by Silverstein et al[76], 24% had Duodenal ulcer, 24.4% had Gastritis/ Erosions, 22.3 % Gastric ulcer, 11.3 % Oesophageal varices, 8.2%Mallory weiss tear, 7.3% Oesophagitis, 6.8% Duodenitis, 2.8% Neoplasms, 1.9% Stomal ulcer.

Based on the clinical variables like age, the presence of shock and the presence of co-morbid illness, the clinical Rockall score was calculated for each patient with non variceal bleeding. The original rockall scoring was calculated by totaling the clinical and endoscopic scoring systems. The scoring results of our set of patients were shown in the Chart.6 and Table.5. It clearly showed that majority of the patients (32) were with the score of 2. Only 5 and 4 patients had the score of 5 and 6 respectively with mortality rate being 4% among them. Thus, it concludes that, higher the rockall score, higher the mortality with significant P value of < 0.001 as shown in the Table.6. The mean Rockall score was 2.4 and 5.5 among the survival and death respectively as shown in the Chart.7. Thus in our study we could stratify patients with a total rockall score of 4 and below as low risk strata and with a score of 5 and above as high risk strata. Rockall scoring has been validated in multiple patient populations across a range of health sittings. It appears to be a valid predictive index that relies on clinical and endoscopic data for assessing the risk of subsequent recurrent bleeding and mortality in patients with acute UGIH.

Patients presented with Upper GI bleeding in emergency ward was prioritized to start initial resuscitation according to the clinical presentation. Peripheral line or Central venous line, Ryles tube insertion and Foley's catheterisation would be started with intense monitoring of

vital signs and urine output. Endotracheal intubation should be attempted in necessary conditions.

Initial fluid resuscitation should be given with crystalloids like Ringer lactate or Normal saline of about 500 ml within the first 30 minutes, followed by colloids like haemacel to maintain the intravascular volume. Compatible cross matched blood transfusion should be started as soon as possible to counteract the blood loss due to haemetemesis. Fresh frozen plasma or Platelets transfusion can also be given depending upon multiple factors, including bleeding severity, bleeding rate, presence of other coagulopathies, and presence of qualitative platelet defects, such as those induced by NSAIDs. Cardio respiratory support, and treatment of significant comorbid diseases, such as sepsis or coronary artery disease must be given importance to prevent morbidity. Patients usually receive supplemental oxygen by face mask or nasal prongs to counteract the loss of oxygen carrying capacity from blood loss. In patients suggestive of Non variceal bleeding, high-dose Proton pump inhibitor therapy i.e, initial intravenous bolus of Pantoprazole 80 mg followed by continuous infusion (Pantoprazole 8 mg/hr) for up to 72 h should be given. This therapy ideally be given prior endoscopy. In our study, 87% of patients were managed with initial fluid resuscitation with crystalloids and colloids, blood transfusion was given in 82% of individuals and 88% of patients had given

proton pump inhibitor therapy as shown in Chart.8. These results are hence comparable with Duggan.J.M et al[46] and Blair.S.D, Janvrin.S.B et al study[47].

Early OGD will significantly improves the clinical outcome in special circumstances requiring urgent endoscopic hemostasis, such as severe, ongoing hemorrhage or esophageal variceal hemorrhage. Bleeding form vaices is mainly because of dysfunction of liver and bleeding varices which are refractive to the treatment. The selection of therapy for bleeding esophageal varices remains controversial. Of all the modes of treatment, in recent years attention was focused on the relative merits of portosystemic shunting, devascularization of esophagus on one hand and the endoscopic procedures to control the bleeding from esophageal varices on the other hand.

The use of endoscopy therapeutically, minimizes the blood transfusion requirements and reduces the length of stay in intensive care set up. Injection therapy can be done over the bleeding site using normal saline or diluted epinephrine. The use of Cautery devices like heater probes, neodymium yttrium aluminum garnet lasers, argon plasma coagulation, and electrocautery probes were used to coagulate blood vessels(coaptation). Clips and band ligation devices were also deployed over the bleeding site

to control it. In our study. therapeutic interventions were needed in 2 patients whose OGD findings showed Gastric polyp and Foreign body. Endoscopic polypectomy and Endoscopic foreign body (needle) extraction were done in the above said cases.

Endoscopic variceal band ligation(EVL) was done in a patient who had presented with Upper GI bleeding and OGD showing Grade III oesophageal varices. The results from 6 randomised control trials showed that, Endoscopic variceal ligation is superior to Endoscopic sclerotherapy(EST)as evidenced from the study conducted by Luketic V.A et al[39] and a meta analysis has confirmed the superiority of EVL over EST by the study of Laine.L Cook.D et al[61]. It was stated that, Endoscopic variceal ligation should be repeated every 2 to 4 weeks to eradicate the varices. Concomitant Beta-blocker therapy should also be considered for effective treatment.

Radiological intervention were required for 2 cases in our study. Angiography along with transcatheter embolisation proved to be valuable as a non operative option for selected patients with non variceal upper gastrointestinal bleding as quoted in a study conducted by Rafique.M.Z et al[71]. Micro coils like, fibred platinum coils of size ranging from 2 – 10 mm in diameter are used. These coils decreases the perfusion pressure thus

allowing the bleeding vessel to get thrombose, once deployed in a distal artery. Coil embolisation of Gastro duodenal artery was done and the patient was improved symptomatically.

In our study, Laparotomy was done for 4 cases as shown in the Chart.9. The operations for bleeding duodenal ulcer are oversewing of the ulcer with or without vagotomy and drainage procedures. Oversewing alone is associated with higher ulcer recurrence but have lower mortality rate. Emergency laparotomy was done for a case of Refractory haemetemis with pharmacological and endoscopic management. OGD finding was duodenal bulb ulcer with visible spurting vessel. Endoscopic banding was failed due to continuous bleeding. Laparotomy was done, under running sutures made over the spurting vessel followed by truncal vagotomy and pyloroplasty and can be compared with the study conducted by de la Fluente.S.G et al[74].

For other three patients with carcinoma stomach, fluid resuscitation and blood transfusion were given initially to stabilize the patient and thorough evaluation was made with investigations. Surgical resection of the growth with adequate margins is the only curative treatment for carcinoma stomach. R0 resection is the goal of surgical treatment. The standard surgery for stomach growth is, Radical sub total gastrectomy, Billroth II

gastrojejunostomy with Roux en Y anastomosis. The operative mortality for this procedure is around 2 to 4 %[77]. Depending upon the per operative findings, procedure can be modified. Palliative procedures like anterior gastrojejunostomy or Feeding jejunostomy can be undertaken in case of locally advanced or metastased gastric carcinoma. In our study , Elective laparotomy was made and one patient with antro pyloric growth underwent subtotal gastrectomy, roux-en-Y anastomosis with D2 resection. Another patient with carcinoma stomach with transverse mesocolon infiltration was managed with palliative anterior gastro jejunostomy, while the elderly patient with advanced carcinoma stomach with peritoneal and liver secondaries underwent feeding jejunostomy.

From the Table.7 and Chart.10, it can be stated that, the mean age of survival among the individuals with upper gastrointestinal bleeding was 44 years and the mortality was seen among the patients with group of around 55 years.

SUMMARY

- This prospective study of Clinical study, Evaluation and Management of Upper gastrointestinal bleeding was done in our institute (Government Stanley Medical College Hospital) on 100 cases, who presented with haemetemesis. The study period was 1 year.

- Upper Gastrointestinal bleeding is potentially a life threatening condition, commonly seen among males than females in the ratio of 89 : 11 in our study.

- The mean age of presentation is 45 years.

- Most of the patients (58%) presented to the emergency ward were with mild haemetemesis (< 500ml).

- The most common clinical presentation is with haemetemesis accompanied with abdomen pain (69%) and malena (45%).

- Association with co-morbid factors, increases the mortality rate.

- Among the risk factors, alcoholism tends to the major one (72%) associated with mortality (75%).

- Gastritis and Erosive gastritis appears to be the commonest OGD finding in our study.

- It was concluded that, Rockall score plays a vital role in predicting the mortality. Higher the rockall score, higher will be the mortaliy.

- Initial fluid resuscitation(87%), Blood transfusion(82%) and Proton pump inhibitor therapy(88%) were given to the individuals depending upon the clinical assessment.

- Intervention procedures were done among 9 individuals in our study. Therapeutic endoscopy in 3 patients, coil embolisation in 2 patients, emergency laparotomy in 1 and elective laparotomy in 3 patients.

- The mean age of mortality is being 55 years in our study.

- Mortality rate in our institute was 4 in 100 cases in our study.

CONCLUSION

- Upper gastrointestinal bleeding is one of the commonest emergencies all over the world. It has an incidence of about 50 to 150 per 100,000 population every year.

- The mean age of presentation of UGIB was 45 years with predominantly seen among males.

- More than half of the patients presented to the emergency ward with mild haemetemesis where as mortality was found to be in individuals with severe haemetemesis.

- Among the risk factors studied, alcoholism appears to be a major risk factor almost always associated with peptic ulcer disease and 3/4 increased risk for mortality.

- The associated co-morbid illnesses plays a significant role in deciding the mortality.

- Oesophago-Gastro-Duodenoscopy was used as a diagnostic and therapeutic tool in the management of UGIB. The most common

finding was Gastritis, followed by Erosive Gastritis. Thus, Peptic ulcer disease is the commonest cause of UGIB.

- The Rockall score was used to triage the non variceal upper gastrointestinal bleed patients into low and high risk strata and it was proved that higher the Rockall score, higher will be the mortality.

- Radiological and surgical interventions should be considered in patients refractory to conservative management, depending upon the circumstances.

- Mortality rate in our institute is 4%, which is best explained by the availability of an Upper GI bleed centre and strict adherence to formed protocols.

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PROFORMA

CLINICAL STUDY, EVALUATION AND MANAGEMENT OF UPPER GASTRO INTESTINAL BLEEDING

Patient details: Patient ID No:…….

Name :

Age/Sex :

IP No :

DOA :

DOP :

DOD :

Address :

Mobile No :

History

Occupation : Rural/Urban

Socio Economic Status : Upper/UpperMiddle/LowerMiddle/Poor

Haemetemesis :

- Onset
- Duration
- Quantity
- Episodes / Frequency
- Association with Guiddiness / Fainting
- Association with Malena

Abdominal Pain

- Onset
- Duration
- Nature
- Severity
- Radiation of pain
- H/o Drug intake

Past History

- H/o Previous episodes
- H/o Previous Surgeries
- H/o DM /HTN/ PT/BA / IHD/ Liver Failure / Malignancies

Personal History

- Alcoholism , If any - Last Bout
- Smoking
- Tobacco / Betel Nut Chewing

General Examination

- Consciousness and orientation
- Pallor
- Jaundice
- Clubbing
- Vital Signs :

  Pulse Rate :
  Blood Pressure :
  Spo2 :
- Ryles Tube Aspirate :

Examination of Abdomen

Per Rectal Examination

CVS :

RS :

CNS :

Investigations:

- Complete blood count
- Renal function tests
- Liver function tests
- Chest X ray PA View
- X ray Abdomen AP erect view
- ECG

Early Resuscitation

- Nasal 02
- Intravenous Crystalloids / Colloids
- Blood Transfusion according to blood Loss
- Empiric Pharmacotherapy

OGD Scopy Findings

Non Variceal

- Oesophagitis
- Mallory - Weiss tear
- Gastric Erosions / Gastritis
- Gastric Ulcer
- Duodenitis
- Duodenal Ulcer
- Neoplasm
- Angiodysplasia
- Dieulafoy lesion
- Miscellaneous

Variceal

- Oesophageal Varices
- Oesophago Gastric Varices

- Gastric varices.

Rockall Risk scoring system	
Variable	Scores
Age (Years)	
< 60 Years 60 - 79 Years > 80 Years	0 1 2
Shock	
Pulse < 100/min, SBP>100 mmHg Pulse > 100/min, SBP>100 mmHg Pulse < 100/min, SBP<100 mmHg	0 1 2
Co-Morbid Conditions	
No Major Co-Morbidity Cardiac Failure, Ischemic Heart Disease Renal Failure, Liver Failure, Disseminated Malignancies	0 2 3
Diagnosis	
Mallory Weiss tear, No Lesion Identified All other Diagnosis Malignancy of Upper GI Tract	0 1 2
Major Stigmata of Recent Haemorrhage	
None / Dark Spot Only Blood in Upper GI Track, Adherent clot, Visible or Spurting Vessel	0 2

Management:

Non -Variceal Causes

- Endoscopic Band Ligation
- Proton Pump Inhibitors
- Anti H-pylori regimen

Variceal Causes :-

Managed in Cooperation with Medical and Surgical Gastro Enterology Department by,

- Endoscopic Sclerotherapy
- Endoscopic Variceal Ligation
- Endoscopic Glue Injection

Follow up

- 24hrs
- 1 week

- 1 Month

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Clinical Study, Evaluation and Management of upper Gastrointestinal Bleeding

Principal Investigator : Dr.K.S. Saravana Krushna Raja

Designation : PG in M.S (Gen.Sur)

Department : Department of Gen.Sur
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 06.08.2012 at the Council Hall, Stanley Medical College, Chen

nai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

30/8/12

MEMBER SECRETARY,
IEC, SMC, CHENNAI

. இரத்த வாந்தியால் பாதிக்கப்பட்டவர்களுக்கு நோய் மற்றும் காரணம் அறிந்து ஆராய்ந்து சிகிச்சை செய்யும் மருத்துவ ஆய்வு

ஆய்வாளர்	:	டாக்டர் எ.எ.சாயல் கிருஷ்ண ராஜ முதுநிலை பட்ட மேற்படிப்பு மாணவர் அரசு சிகிச்சை பட்டப்படிப்பு
வழிகாட்டி	:	பேராசிரியர்.டாக்டர்.ஆ.ராஜேந்திரன் அரசு சிகிச்சை நோயியல் அரச ஸ்டான்லி மருத்துவமனை

பங்கேற்பாளரின் நலவல் படிவம்

நீங்கள் இந்த ஆய்வில் பங்கேற்க அழைக்கப்படுகிறீர்கள்.

இந்த ஆய்வில் பங்கேற்கும் முன்னர் இதன் நோக்கத்தையும் முறைகளையும் இதனால் ஏற்படக்கூடிய பின் விளைவுகள் ஏதேனையும் நீங்கள் அறிந்து கொள்ள ஆய்வாளர் அனைத்தும் நலவல் பின்வருமாறு.
இந்த வாந்தியால் பாதிக்கப்பட்ட நோயாளிகள் மட்டுமே இந்த ஆய்வில் எடுத்துக் கொள்ளப்படுவீர்கள். உங்கள் நோயின் முழு வரலாறும் உங்களின் முழு உடல் பரிசோதனையும் நெஞ்சகமும் வீரோகமும் பதிவு செய்யப்படும். உங்கள் நோய் கண்டுபிடிக்கத் தேவையான மருத்துவபரிசோதனைகள் மேற்கொள்ளப்படும்.

உணவுக்குழாய் உள்நோக்கி மூலம் இரத்த வாந்திக்கான காரணம் கண்டறியப்படும். ரத்தம் எடுக்கும் மூலமாக நோயின் தீவிரத்தையும் அபாயத்தையும் அறிந்து உடனடியாக பின்பற்றப்படும், மேலும் தங்களுக்கு தேவையான வினைகிச்சையும் சிகிச்சை முறையே செய்யப்படும்.
இவ்வாய்வினால் தங்களுக்கு எந்த விதமான பாதிப்பும் ஆபத்தோ ஏற்படாது. ஆனால் தங்கள் நோயினையோ அதற்குரிய பரிசோதனைகளையோ தங்கள் நோய்குரிய அறுவை சிகிச்சையினையோ அதன் பின்னர் ஏற்பட வாய்ப்புள்ள சிக்கல்களினையோ ஏற்படக்கூடிய ஆபத்துக்களுக்கும் இந்த ஆய்வுக்கும் எந்த சம்பந்தமும் கிடையாது.

இந்த ஆய்வில் முடிவுகள் மருத்துவக் காரணங்களுக்காகவும் மருத்துவக் கல்விக்காகவும் பயன்படுத்தப்படும். இந்த ஆய்வு பற்றிய எந்தேகங்களுக்கு உரிய முறையில் விளக்கமளிக்கப்படும். தங்களைப் பற்றிய தகவல்கள் ரகசியமாகப் பாதுகாக்கப்படும்.

இந்த ஆய்விலிருந்து எப்பொழுது வேண்டுமானாலும் தாங்கள் எவ்வித முன்னறிவிப்பின்றியும் எவ்வித சட்ட சிக்கலும் இன்றியும் விலகிக் கொள்ளலாம்.

இந்த ஆய்வில் பங்கேற்றமைக்கு நன்றி கொள்கிறோம்.

நன்றி

ஆய்வாளர் கையொப்பம்	நோயாளியின் கையொப்பம்
(டாக்டர்.எ.எ.சாயல கிருஷ்ண ராஜா)	பெயர் :

இரத்தவாந்தியால் பாதிக்கப்பட்டவர்களுக்கு நோய் மற்றும் காரணம் அறிந்து ஆராய்ந்து சிகிச்சை செய்யும் மருத்துவ ஆய்வு

ஆராய்ச்சி இலையம் : அரசு ஸ்டான்லி மருத்துவமனை
சென்னை – 600 001.

பங்கு பெறும் நோயாளியின் பெயர் : வயது :

பங்கு பெறும் நோயாளியின் எண் : பாலினம் : ஆண் ☐ பெண் ☐

நோயாளியின் விலாசம் :

நோயாளி இதனை (✓) குறிக்கவும்.

மேலே குறிப்பிடப்பட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும். அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் என்னை இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்க அனுமதிக்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் என்னை இவ்வாய்வில் இருந்து விலக்கி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்பந்தமாகவோ, இந்த சார்ந்த தேறும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். என்னை ஆய்வில் இருந்து விலக்கி கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்குக் கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி, நடந்துக் கொள்ளுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அலுக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ அதை உடனே மருத்துவ அலுவிடம் தெரிவிப்பேன் என உறுதியளிக்கிறேன். ☐

இந்த ஆய்வில், எனக்கு இரத்தம், சுக்ஸே, இ.சி.ஜி., எக்கோ, பரிசோதனை செய்துக்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன். ☐

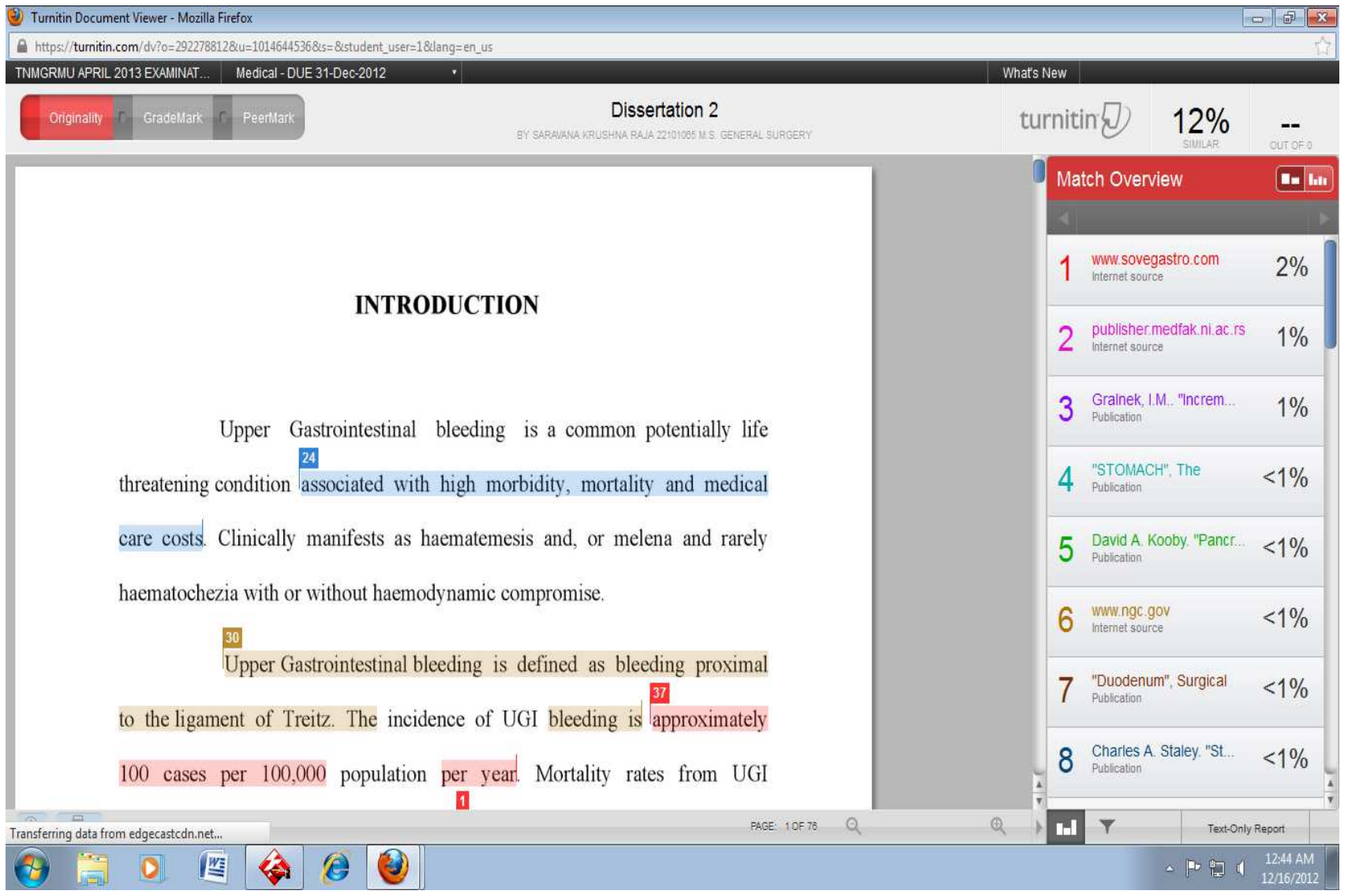
நோயாளியின் கையொப்பம் ..... இடம் ..... தேதி

கட்டைவிரல் ரேகை (இந்த படிவம் படித்து காட்டப்பட்டு புரிந்து கையொப்ப அளிக்கின்றேன்)

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர் .....

-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INTRODUCTION
Upper Gastrointestinal bleeding is a common potentially life threatening condition associated with high morbidity, mortality and medical care costs. Clinically manifests as haematemesis and, or melena and rarely haematochezia with or without haemodynamic compromise.
Upper Gastrointestinal bleeding is defined as bleeding proximal to the ligament of Treitz. The incidence of UGI bleeding is approximately 100 cases per 100,000 population per year. Mortality rates from UGI
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MASTER CHART

NAME	IP.NO	AGE	SEX	ALCOHOLISM	SMOKING	NSAIDS	OGD FINDINGS	ROCKALL SCORE	RESUSCITATION	BLO
MARIYAPPAN	13612	35	M	NO	YES	NO	GASTRIC POLYP	1	NO	N
YASODHA	13716	40	F	NO	NO	YES	NORMAL STUDY	0	NO	N
MURUGAN	25132	30	M	YES	YES	NO	EROSIVE GASTRITIS	2	YES	YE
RATHINAVEL	25612	42	M	YES	NO	NO	MALLORY WEISS TEAR	3	YES	YE
MOHAMMAD ALI	13176	60	M	YES	YES	NO	OESOPHAGITIS	5	YES	N
RAMASAMY	27765	70	M	YES	YES	NO	DUODENAL ULCER	2	YES	YE
VENKATESAN	24129	60	M	NO	YES	NO	OESOPHAGEAL VARICES	6	YES	YE
MANI	26172	38	M	YES	NO	NO	MALLORY WEISS TEAR	1	YES	N
ANVAR BASHA	28861	62	M	YES	NO	NO	OESOPHAGITIS	2	YES	N
MUNUSAMY	29618	52	M	YES	YES	NO	GASTRITIS	5	YES	YE
DHARMANDRAN	25001	35	M	YES	YES	NO	DUODENAL ULCER WITH CLOT	4	YES	YE
PATCHAIPPAN	24126	58	M	YES	NO	NO	OESOPHAGEAL EROSION	3	YES	N
LOGANATHAN	27111	60	M	YES	YES	NO	DUODENAL ULCER	4	YES	YE
ALAMELU	27574	42	M	YES	YES	NO	GASTRITIS	1	YES	YE
MANICKAM	28112	62	M	YES	YES	NO	OESOPHAGITIS & GASTRITIS	1	YES	YE
NADEEM BASHA	28612	78	M	YES	YES	NO	LESSER CURVATURE GROWTH	6	YES	YE
RAMAMOORTHY	30061	56	M	YES	YES	NO	EROSIVE GASTRITIS	3	YES	YE
NAINA	30965	18	M	NO	YES	YES	DUODENAL ULCER WITH BLEED	4	YES	YE
DEEPAK JALOB	31216	49	M	YES	YES	NO	GASTRITIS & DUODENITIS	3	YES	YE
MANNAR	31132	44	M	YES	NO	NO	EROSIVE GASTRITIS	1	YES	YE
KADHAR BASHA	31218	52	M	YES	NO	NO	DUODENITIS	3	YES	YE
GANGADHARAN	31269	38	M	YES	YES	NO	OESOPHAGITIS & GASTRITIS	1	YES	YE
ARUNACHALAM	31137	40	M	NO	YES	YES	GASTRITIS	2	YES	YE
MANICKAM	26172	64	M	YES	YES	NO	EROSIVE GASTRITIS	5	YES	YE
MUTHUMANI	29016	48	F	NO	NO	YES	GASTRITIS	2	YES	YE
BASKARAN	31109	34	M	NO	YES	NO	NORMAL STUDY	1	YES	YE
ILANGOVAN	31122	44	M	YES	YES	NO	EROSIVE GASTRITIS	1	YES	YE
MOHAN RAJ	31061	38	M	YES	NO	NO	GASTRITIS	1	YES	YE

JAYA RAMAN	32161	53	M	YES	YES	NO	DUODENAL ULCER WITH CLOT	3	YES	YE
THANGAVEL	31763	24	M	YES	NO	NO	GASTRITIS	2	YES	YE
NATESAN	30176	64	M	YES	NO	NO	OESOPHAGEAL VARICES	4	YES	YE
CHAKKARAPANI	31619	37	M	NO	NO	YES	OESOPHAGITIS & LAX LES	3	YES	YE
MANJULA	31619	35	F	NO	NO	NO	OESOPHAGITIS	1	YES	N
SARGUNAM	30179	45	M	YES	YES	NO	GASTRIC ULCER	2	YES	YE
RAJENDRAN	30888	59	M	YES	NO	NO	GASTRITIS & DUODENITIS	3	YES	YE
VASANTHA KUMAR	31767	28	M	YES	NO	NO	GASTRITIS	2	YES	YE
CHANDRAN	31212	25	M	YES	NO	NO	DUODENITIS	1	YES	YE
NAGARAJAN	32617	34	M	YES	YES	NO	EROSIVE GASTRITIS	2	YES	YE
RAMESH	31769	36	M	NO	YES	YES	OESOPHAGITIS	1	YES	N
MUTHU	32361	78	M	YES	YES	NO	DUODENAL ULCER & LAX LES	3	YES	YE
PALAYAM	33744	64	M	YES	YES	NO	GASTRITIS	2	YES	YE
GURUMOORTHY	30921	42	M	YES	NO	YES	GASTRIC ULCER WITH CLOT	4	YES	YE
RAMADOSS	30612	54	M	YES	NO	NO	EROSIVE GASTRITIS	3	YES	YE
GANGADHARAN	30717	36	M	NO	NO	YES	STOMAL ULCER	4	YES	YE
REICHEL	30597	45	F	NO	NO	YES	GASTRITIS	2	YES	YE
PALANI	39181	40	M	NO	YES	NO	LESSER CURVATURE GROWTH	3	NO	YE
ARUN PRAKASH	37165	24	M	YES	NO	NO	GASTRITIS	2	YES	N
NARASIMMALU	36543	49	M	YES	YES	NO	GASTRITIS	3	YES	YE
RAJA	37754	33	M	NO	YES	YES	GASTRITIS	2	YES	YE
MANOHARAN	35784	54	M	YES	NO	YES	GASTRIC ULCER	3	YES	YE
RAMARAJU	38675	40	M	YES	YES	NO	DUODENAL ULCER WITH BLEED	5	YES	YE
ANGAMUTHU	39687	50	M	YES	YES	NO	OESOPHAGITIS & GERD	3	YES	N
SARAVANAN	40124	23	M	NO	NO	NO	FOREIGN BODY - NEEDLE	0	NO	N
VIGNESWARAN	41221	35	M	YES	NO	NO	GASTRITIS	2	YES	YE
RAMAYEE	41576	52	F	NO	NO	YES	GASTRITIS	2	YES	YE
KUMARAN	40582	38	M	NO	YES	YES	GASTRIC VARICES	2	YES	YE
ALBERT	40985	39	M	YES	YES	NO	MALLORY WEISS TEAR	1	YES	YE
BALA MURUGAN	40791	42	M	NO	YES	NO	GASTRITIS	2	YES	YE
JAMES	41005	50	M	YES	YES	NO	EROSIVE GASTRITIS	3	YES	YE
SELVAM	41082	36	M	YES	YES	NO	DUODENITIS	3	YES	YE
RAMACHANDRAN	42001	29	M	YES	NO	NO	GASRRITIS & DUODENITIS	3	YES	YE
MUNIYAMMAL	42975	79	F	NO	NO	YES	EROSIVE GASTRITIS	4	YES	YE
PRATAP	42912	33	M	YES	YES	NO	GASTRITIS	2	YES	YE

KANDASAMY	42765	46	M	YES	YES	NO	EROSIVE GASTRITIS	2	YES	YE
SADHASIVAM	42217	55	M	YES	YES	NO	GASTRITIS	3	YES	YE
PARIMALA	42665	35	F	NO	NO	YES	GASTRITIS	1	NO	N
SENTHIL	43167	40	M	YES	NO	NO	DUODENITIS	1	NO	YE
KUMARAN	43288	42	M	YES	YES	NO	OESOPHAGITIS	1	YES	YE
RAMANAN	43312	48	M	NO	NO	YES	GASTRITIS	2	NO	YE
SARAVANAN	43589	24	M	YES	NO	NO	DUODENITIS	2	YES	YE
ASHIQ	44645	34	M	YES	NO	NO	MALLORY WEISS TEAR	1	NO	N
PARTHASARATHY	45623	40	M	YES	YES	NO	EROSIVE GASTRITIS	2	YES	YE
SIVA MURUGAN	45678	70	M	YES	NO	NO	LESSER CURVATURE GROWTH	3	YES	N
MICHAEL	46685	62	M	YES	YES	NO	GASTRITIS	3	YES	YE
HUSSAIN ALI	46675	54	M	YES	NO	NO	LOWER OESOPHAGEAL VARICES	2	YES	YE
BAKIYARAJ	46754	34	M	NO	NO	NO	OESOPHAGITIS	2	YES	N
THIRU MOORTHY	46864	28	M	YES	YES	NO	DUODENITIS	2	YES	YE
KESAVAN	47687	35	M	YES	NO	NO	OESOPHAGITIS	2	NO	YE
RAMU	47987	75	M	YES	YES	NO	DUODENITIS	3	YES	YE
MALARVIZHI	46574	40	F	NO	NO	YES	GASTRITIS	3	YES	YE
RAMESH	47275	35	M	YES	YES	NO	DUODENAL ULCER	3	YES	YE
YOUNIS KHAN	48124	42	M	YES	NO	NO	NORMAL	2	NO	N
BOOBALAN	48235	37	M	YES	YES	NO	GASTRITIS	3	YES	YE
PARANTHAMAN	48432	38	M	NO	NO	NO	OESOPHAGITIS	2	NO	N
BALARAMAN	48356	45	M	YES	YES	NO	EROSIVE GASTRITIS	3	YES	YE
PARAMASIVAM	47265	38	M	YES	YES	NO	MALLORY WEISS TEAR	1	NO	YE
SATHISH	48734	35	M	YES	YES	NO	GASTRITIS	2	YES	YE
MUNUSAMY	49947	42	M	YES	YES	NO	EROSIVE GASTRITIS & LAX LES	2	YES	YE